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U.S. NAVAL

Flight Surgeon's Manual

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Physiological Psychology Branch
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UNDER THE AUSPICES OF
THE CHIEF OF NAVAL OPERATIONS AND
THE BUREAU OF MEDICINE AND SURGERY
DEPARTMENT OF THE NAVY

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Foreword

Since the very beginning of the U.S. Navy, the medical profession has played its part. Medical officers have served on every type of ship, beginning with the *Alfred,* the vessel on which John Paul Jones hoisted the first American flag in 1775. Initially, medical officers were given no special training but were simply signed on as members of the ship's company for a particular voyage. Indeed, for years no special training was required since the responsibilities of a medical officer aboard ship were essentially no different from those of one treating a civilian population. However, as naval operations became more specialized, the need for unique medical skills became apparent. This has been especially true in naval aviation, where the aviator is subjected to a multitude of unusual stresses. If a Flight Surgeon is to insure the well-being of aviation personnel, he must fully understand the aerospace environment and the physical and psychological problems which may result from operating within this environment. Aerospace medicine has indeed become a specialized medical science.

This manual represents a continuing effort by the Bureau of Medicine and Surgery to provide the latest information concerning human response to the aerospace environment and to describe the occupational requirements of the Flight Surgeon. Over the years, Flight Surgeons have made a notable contribution to naval aviation and to our general fund of information within the medical sciences. I am pleased to be given the opportunity to dedicate this manual to those who will continue this tradition.

Without the assistance of Commander Channing L. Ewing, MC, USN, who acted as our Project Officer and Scientific Editor, this manual would not have been possible. He contributed substantially through his efforts during the organization, preparation, and review of all materials. A special word of appreciation is due Captain Marvin D. Courtney, MC, USN, who has guided this manual through to its publication.

W. M. Snowden
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Assistant Chief for Aerospace Medicine
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Navy Department
Washington, D.C. 20390

Preface

This manual is directed toward the specialized practice of naval aerospace medicine. The purpose is to describe the specific activities of a Navy Flight Surgeon and to present content information required by the Flight Surgeon as he performs these activities.

Much of the information contained in this manual was obtained during visits to the following facilities:

The Office of the Assistant Chief for Aerospace Medicine
Bureau of Medicine and Surgery
Navy Department
Washington, D.C. 20390

The Faculty of the Naval Aerospace Medical Institute
Naval Air Station
Pensacola, Fla. 32512

Medical Officer
Commander, Naval Air Force, U.S. Atlantic Fleet
Naval Air Station
Norfolk, Va. 23511

Naval Aviation Safety Center
Naval Air Station
Norfolk, Va. 23511

The Office of the Assistant Chief for Dentistry
Bureau of Medicine and Surgery
Navy Department
Washington, D.C. 20390

Crew Systems Division
Naval Air Systems Command
Navy Department
Washington, D.C. 20360

Aerospace Crew Equipment Department
Naval Air Development Center
Johnsville, Warminster, Pa. 18974

Aerospace Medical Research Department
Naval Air Development Center
Johnsville, Warminster, Pa. 18974

Medical Department
USS *America* (CVA-66)

Navy Preventive Medicine Unit No. 2
Naval Air Station
Norfolk, Va. 23511

Staff Medical Office
Headquarters, Marine Corps
Washington, D.C. 20380

Second Marine Aircraft Wing
Marine Corps Air Station
Cherry Point, N.C. 28533

Armed Forces Institute of Pathology
Washington, D.C. 20012

Biotechnology and Human Research Division
Office of Advanced Research and Technology
Headquarters, National Aeronautics and
Space Administration
Washington, D.C. 20546

Without exception, persons visited at these facilities were most helpful in providing content information and useful suggestions for the structure of the manual.

Peggy Morgan Padlar served as Editor during the preparation and coordination of these diverse materials for publication. Her outstanding contribution is gratefully acknowledged.

JAMES F. PARKER, Jr.
BioTechnology, Inc.

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CHAPTER 1

A History of Aerospace Medicine in the U.S. Navy

LCDR Warren Hodge, MC, USN

THE HISTORY of aerospace medicine in the U.S. Navy is intimately interwoven into the growth of aviation and of naval aviation. In 1909, only six years after the first aircraft flight of the Wright brothers, LT George C. Sweet became the first naval officer to fly.

In 1910, CAPT I. Chambers was assigned by the Secretary of the Navy to oversee aviation matters of possible interest to the Navy. In the autumn of 1910, CAPT Chambers and Glenn Curtiss met at air meets where they discussed the practicality of aircraft for Navy use aboard ships. A Curtiss pilot, Eugene Ely, agreed to make the attempt. On 14 November 1910, he flew a four-cylinder Curtiss biplane from a wooden platform built on the deck of the USS *Birmingham*. On 18 January 1911, Eugene Ely topped this feat by landing his aircraft on board a makeshift flight deck on the armored cruiser USS *Pennsylvania,* then turning his plane about and flying back to shore. For the landing aboard ship, he was arrested by transverse cables attached to sandbags. Although these demonstrations were convincing, the Secretary of the Navy, George von L. Meyer, wrote to Curtiss that he was waiting for an aircraft that could land in the water alongside a ship and be hoisted aboard. On 17 February 1911, the inventor himself landed his aircraft, equipped with floats, beside the *Pennsylvania* and was hoisted aboard by the boat crane. An interested spectator at these proceedings was T. G. Ellyson, who had been ordered to the Curtiss Camp at North Island, Calif., for flight instruction, with orders dated 23 December 1910. These were the first orders to flight instruction, thus making Lieutenant Ellyson Naval

Aviator Number One. By the end of 1912, eight other officers had joined Ellyson as naval aviators. Naval aviation had begun in earnest.

In 1912, the first set of medical standards in this country for aviation candidates was published by the War Department. On 8 October 1912, the Navy Bureau of Medicine and Surgery issued the first set of physical requirements for naval candidates for aviation duty.

In January 1913, the small group of naval aviators joined the Fleet for winter maneuvers at Guantanamo to demonstrate the operational abilities of the aircraft and to give flights to interested officers and personnel. In January 1914, naval aviation was given considerable impetus by the statement from the Secretary of the Navy that, "The science of aeronavigation has reached that point where aircraft must form a large part of our naval force for offensive and defensive operation." At that very moment, two ships, the USS *Mississippi* and the USS *Orion,* were on their way to the Navy Yard at Pensacola, Fla. On 20 January 1914, they arrived and began offloading their cargo of aircraft and supporting gear. This was the beginning of the Naval Aeronautics Station at Pensacola, later to become the home of the U.S. Naval Aerospace Medical Center.

In 1916, Col. Ralph Green, USA, became the first medical officer ordered to flying duty. In that same year, Maj. Theodore Lyster, Chief of Aviation Medicine and Professional Services of the U.S. Army Medical Department, with assistance from Colonels Wilmer and Jones, devised a new set of aviation standards. With the great expansion of flight training as the United States entered the war in 1917, Lt. Col. Lyster was placed in charge of physical examinations for trainees and assumed staff duty as the first Chief Surgeon of the Signal Corps. Because of his early interest and work in aviation medicine, Dr. Lyster has been called "the father of aviation medicine in the United States."

In 1917, an Aviation Medical Research Board was appointed by the Army. Among its acts was the establishment of the Air Service Central Medical Research Laboratory, in January 1918, at Hazelhurst Field, Long Island, N.Y. Dr. Lyster and other Army medical officers had visited the Continent to observe the medical support of aviation units, first hand, in December 1917, and returned to implement their findings. Research into medical problems and some training of medical officers was undertaken. In August 1918, a group from the Central Research Laboratory established a research laboratory in France for the study of aviation problems and support of American

Expeditionary air units. This effort had dramatic results in reducing flying accidents and deaths.

In May 1919, a new section was established in the Army's Central Research Laboratory. Called a School for Flight Surgeons, this section offered the first formal course in aviation medicine. The Research Laboratory and School for Flight Surgeons were moved to nearby Mitchell Field in November 1919. After a disastrous fire in 1921, the Research Laboratory was disbanded. The School was rebuilt and officially designated the School of Aviation Medicine on 8 November 1922.

In 1921, the Navy began sending medical officers to be trained at the U.S. Army School of Aviation Medicine at Mitchell Field. The first class of five officers graduated on 29 April 1922. They were LTs Victor S. Armstrong, Louis Iverson, Julius F. Newberger, Page O. Northington, and Carl J. Robertson. Their instructors included the Commandant, Louis H. Bauer, and other aviation medicine pioneers such as Edward Schneider and Raymond Longacre.

Soon after LT Armstrong left the School, he was ordered to the Bureau of Medicine and Surgery to become its first Chief of the Aviation Medicine Division in 1923. Naval Flight Surgeons continued to be trained at the Army school through 1926. From 1927 to 1936, Flight Surgeons were trained at the Navy Medical School at Bethesda, under a course of instruction devised by Dr. Armstrong and others. The Army School was moved to Brooks Field, San Antonio, Tex., in 1926, and in 1931, to nearby Randolph Field.

Navy medical officers received several months of practical training at the Naval Air Station, Pensacola, following their graduation as Flight Surgeons. In 1922, LT Bertram Groesbeck Jr., became the first naval medical officer to take flight training and to receive his wings as a Naval Aviator. He then was ordered to the Army School, where he graduated as a Flight Surgeon with the third Navy class in April 1923. A small number of other Flight Surgeons also completed flight training and became naval aviators.

The Flight Surgeon of the 1920s often had duty with organizations other than those at an aviation facility, and continued to have no extra pay and no special insignia. The career of John R. Poppen is an example of activity during this period between wars. He was a graduate of the second class of Navy Flight Surgeons in 1922. From 1926 to 1928, he taught Aviation Medicine at the Navy Medical School, Bethesda. He was one of the charter members of the Aeromedical Association, along with Louis H. Bauer and others in 1929. In 1931, he began work at the Harvard School of Public Health in the physiologic effects of high acceleration. In 1933, he was transferred to the Naval Aircraft Factory at Philadelphia, where he continued work in the development of an antiacceleration belt, the forerunner of the anti-G suit. He was instrumental in the development of the anti-G suit while assigned to the Medical Research Section of the Bureau of Aeronautics in the early 1940s.

Another early Flight Surgeon, Frederick Ceres, became the first Navy medical officer to make a parachute jump, when he jumped with a group of six men and six officers in 1929. He also was a founding member of the Aeromedical Association and a graduate of the third class of Flight Surgeons at Mitchell Field.

In 1936, the Aeromedical Association met in Los Angeles. The first day's session was held aboard the carrier, USS *Lexington*. CDR John R. Poppen gave a presentation on "Equilibratory Functions in Instrument Flying." In all, ten presentations were given, the most ambitious scientific session attempted up to that time. Navy Flight Surgeons have continued to be very active in this organization to the present ime.

In 1936, naval medical officers were again sent to the Army School of Aviation Medicine, then located at Randolph Field in Texas. As before, these Flight Surgeons, after graduation, spent several months at Pensacola for practical training. Through 1939, 56 Navy Flight Surgeons had been graduated from the Army School, and another 25 had been trained at the Navy Medical School, Bethesda, during 1927 to 1936. At this time, serious discussion began concerning the possibility of the Navy establishing its own school for Flight Surgeon training. CAPT J. C. Adams, who was Head of the Aviation Medicine Division of the Bureau of Medicine and Surgery, CAPT Frederick Ceres, who was the Medical Officer at the Naval Air Station, Pensacola, and others were quite interested in a separate training program for Navy Flight Surgeons.

On 8 November 1939, President Franklin D. Roosevelt declared a limited state of emergency. On 20 November 1939, nine Reserve medical officers reported for training in aviation medicine to the Medical Department of the Naval Air Station, Pensacola, Fla. CAPT Frederick Ceres was the Medical Officer at that time and was given the duty of organizing a training course and administering the program. Only a few days' notice had been given of the arrival of these officers for training. Classes first were held in the dispensary building, and in December 1939, were moved to the new Medical Department. The first faculty consisted of CAPT Frederick Ceres; CAPT Alfred W. Chandler, DC; CDR Victor S. Armstrong; CDR Frank E. Tierney; LCDR William W. Davies; LCDR Rex H. White (Retired); LCDR Wilbur E. Kellum; LCDR Anselm C. Hohn; and LT Julian Jordan. The syllabus for that first class consisted of 60 days of instruction. The subjects and instructors were:

Cardiovascular Disease	CAPT Ceres.
Neuropsychiatry	CDR Armstrong, LCDR White, LCDR Kellum.
General Examination	LCDR Davies.
Eye, Ear, Nose, and Throat	LCDR Hohn and LT Jordan.
Dental Examination	CAPT Chandler.

The first class of the Navy School of Aviation Medicine graduated on 20 January 1940, and its members were designated as Aviation Medical Examiners. It was the policy in these first years at the school to give 60 days of training to the Reserve officers and give them the designation of Aviation Medical Examiners. Regular medical officers were given a Phase I, which consisted of ground school and flight instruction of 60 days. This was followed by Phase II, 60 days of didactic work which was identical to the Reserve course and which was taken with a Reserve officer class. A final Phase III consisted of six weeks of additional duty at the outlying fields and the Aviation Examining Room, working under the direction of more experienced Flight Surgeons. Regular officers were designated as Naval Flight Surgeons after completing this course. The first group of five regular officers graduated on 30 November 1940.

The school became involved in research early in its existence. In 1940, a contract was let to the Harvard Fatigue Laboratory for preliminary studies on the selection and qualifications of aviators. A Harvard Research Group came to Pensacola in the summer of 1940 to conduct studies on aviation students and instructors. This group was sponsored by the Civil Aeronautics Authority and the National Research Council, as well as by the U.S. Navy. Intensive work was carried out during July, August, and September and continued on a reduced scale until May 1941. A total of 1,056 students and instructors were put through rather complete physiologic studies, psychologic studies, electrocardiograms, electroencephalograms, somatotyping, and cardiac workups. This was the origin of the now famous "1,000 Aviator" study which has been followed up at intervals through the years.

Civilian scientists and doctors who had been interested in aviation medicine began working with the Navy. Ross McFarland from Harvard, who had pioneered the medical support of Pan American Airways and had done much physiological research into effects of altitude as well as work in pilot-selection criteria, was commissioned in the USNR and was a member of the Harvard study group. Eric J. Liljencrantz, who had been head of Pan American's San Francisco medical facility, came on active duty as a lieutenant commander and continued research work. Ashton Graybiel, already a well-known cardiologist, was commissioned a lieutenant commander in the USNR in 1940 and worked with the Harvard Fatigue Laboratory and the Harvard study group at Pensacola. He came to active duty in 1942 and joined the faculty of the School of Aviation Medicine in that year.

Construction had begun in 1940 on a new building for the expanding school. This was completed in 1941 and dedicated on 24 February 1941. Also in 1941, the first physiological training equipment and low pressure chambers were set up and began operation. LCDR Chalmers L. Gemmill came on active duty from his work as Associate Professor of Physiology at the Johns Hopkins University School of Medicine to take charge of this physiological training unit and the low pressure chamber operations. The chambers were used to indoctrinate flight students concerning the hazards associated with ascent to altitude and also on the use of oxygen equipment. A standard indoctrination syllabus was set up and became a part of each flight student's training.

With the onset of World War II, the training program was accelerated with larger classes of Flight Surgeons, and classes convening more frequently. In the Pacific, the tactics of naval aviation and carrier strike forces were utilized to their fullest extent. The well-trained unit Flight Surgeon proved indispensable in keeping flyers healthy and fit to perform their arduous duties.

Back at the school, new members joined the faculty and research staff. LCDR Ashton Graybiel took the academic course and was designated an Aviation Medical Examiner. He later was designated a Naval Flight Surgeon. LT J. L. Lilienthal joined the staff from the Johns Hopkins University School of Medicine and worked on the effects of reduced barometric pressure, carbon monoxide, and airsickness, and in selection criteria for aviators.

The curriculum of the school gradually changed in keeping with the emphasis on support of operational units. With the original idea of creating aviation medical examiners for the cadet selection boards, the original course had stressed physical examination and selection. Now emphasis was also placed on operational aviation medicine, maintenance of pilot health, survival, air evacuation, and many other subjects.

In 1942, provision was made for the Aviation Medical Examiner to be able to change his designation to Naval Flight Surgeon. The requirements were that he complete 6 months with an operational unit after graduation, that he accumulate 60 hours of flying time, and that he have the recommendation of his senior Medical Officer. Most of these early Aviation Medical Examiners fulfilled these requirements and were designated Naval Flight Surgeons.

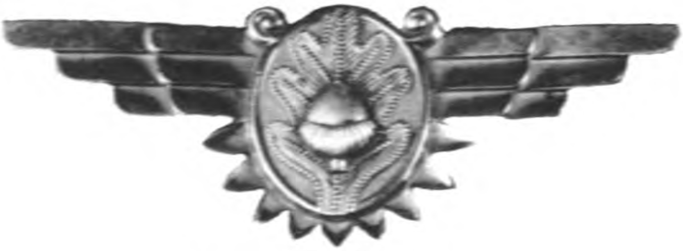
On 18 May 1942, the Chief of Naval Personnel authorized the first Flight Surgeon insignia, "to be worn on the left breast by officers of the Medical Corps who are qualified as Naval Flight Surgeons." The insignia was a metal pin, winged, with an ovoid central device surcharged with the gold leaf and silver acorn insignia of the Medical Corps. There was a fringe below the central device and three horizontal wing feathers on each side. The first set of these wings was hurriedly fabricated by the Dental Department at the Naval Air Station, Pensacola. Figure 1–1 shows the first wings, as well as the current version, and the Medical Corps insignia worn by Flight Surgeons.

The first set of wings was presented to CAPT Frederick Ceres on 19 July 1942, by the Commandant of the Naval Air Station, Pensacola, CAPT A.C. Read, at a farewell party after his relief as first Medical Officer in charge of the School of Aviation Medicine. His relief, as second officer in charge of the school, was CAPT Bertram Groesbeck, Jr.

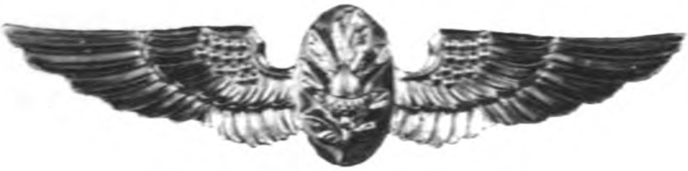
Naval Flight Surgeons now had an insignia of their designation. On 10 October 1942, the Secretary of the Navy sent an amended definition of "nonflying" officer to the Chief of the Bureau of Supplies and Accounts. In it, Naval Flight Surgeons were, for the first time, administratively included under the term "flying officers" and were entitled to draw flight pay while detailed to duty involving flying.

On 5 November 1942, CDR Eric Liljencrantz was killed near the Naval Air Station, Pensacola, while engaged in an experimental flight testing a new accelerometer. This accident cut short his productive and promising career before his 40th birthday. During his service with the Navy from 1940, he had published several papers, given presentations at the Aeromedical Association, and had worked in aviation medicine research.

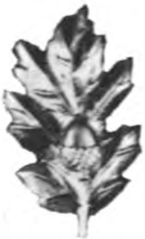
At the 1942 meeting of the Aeromedical Association, CAPT John R. Poppen was elected president of the association. He was the first of six Naval Flight



First Flight Surgeon Insignia



Present Flight Surgeon Insignia



Medical Corps Insignia

FIGURE 1–1. *Insignia worn by Navy Flight Surgeons.*

Surgeons to hold that office through 1962, and was the first military Flight Surgeon to hold that office. The Aeromedical Association continued to have the active participation of naval aviation medicine during the war years as well as before and after the war. Presentations were made by Navy Flight Surgeons and scientists at the annual meetings. Members of the service and staff of the school attended these sessions in great numbers. Papers were submitted and published in the Association's publication, *The Journal of Aviation Medicine.*

By 1 January 1943, the school had acquired three new buildings. One was given over to research, one housed the three low pressure chambers, and the third held classroom and training spaces. This was badly needed space as the Flight Surgeon classes had increased in numbers to 40 or more per class, and a new class was convening every six weeks. Research was continuing and branching into new fields.

By 1944, over 1,000 doctors had graduated from the School of Aviation Medicine. The library had increased in size to 800 books, 50 periodicals, and over 1,000 reprints. Research work was continuing apace. The Research Department was enlarged by the completion of an acceleration unit and the human centrifuge in July 1944. Testing of the equipment was begun and almost immediately was marred by tragedy. LT Austin H. Osgood, a line physicist, was killed on 22 August 1944, while operating the centrifuge on a demonstration run. The installation and use of the equipment continued under his successors, after a delay while improvements were made in the safety design. Research into speech intelligibility and communication was undertaken. Work began concerning problems of pilot disorientation and its relationship to acceleration.

By the end of World War II, the School of Aviation Medicine had graduated 42 classes of trainees. Over 1,500 Flight Surgeons had been designated during this period. Twenty-seven Flight Surgeons had been killed in enemy action or in aircraft accidents during the war. The death rate of Flight Surgeons as opposed to regular Navy medical officers was almost three times as great. Devotion to duty and heroism were the rule rather than the exception. Many Flight Surgeons were decorated for individual acts. Many fought alongside their men in the air and on the ground.

Through 1945, 21 Naval Flight Surgeons had also received the designation of Naval Aviator after completing flight training. A small number of Flight Surgeons were approved each year for completion of full flight training.

During World War II, the training of enlisted medical personnel as Aviation Medical Technicians and as personnel to assist in low pressure chamber operations was begun. After the war, the School of Aviation Medicine became the sole place authorized to train these technicians and has continued to do so. These technicians were and are invaluable assistants to the Flight Surgeon in his work in the field.

At the end of World War II, the public reaction of the United States was one of relief and a desire to "get back to normal." For the armed services, this meant a rapid and drastic cutback to a peacetime level. Naval aviation medicine felt this cut as Flight Surgeons were released from active duty, training programs were diminished, and even research was curtailed. Flight Surgeon Class No. 46, convened in September 1946, contained but 13 medical officers. All of these were allowed to take flight indoctrination.

On 15 October 1946, the school was officially designated by the Secretary of the Navy as the U.S. Naval School of Aviation Medicine and Research. Up to this time, the school had not had official recognition and had developed as an adjunct to the Station Medical Department, with the Senior Medical Officer as Officer-in-Charge. After this, it was a separate activity under the Naval Air Training Command and the Naval Air Basic Training Command with a medical officer as Officer-in-Charge. The first Officer-in-Charge was CAPT Louis Iverson.

At the School of Aviation Medicine, demobilization cut into the research program drastically. The efforts of CAPT Ashton Graybiel, who became Director of Research, kept the Research Department functioning. Plans were made to have certain universities perform research under grants from the Office of Naval Research. The idea of establishing permanent civil service positions in research at the school was conceived and implemented. Recruitment of scientists to fill these positions went forth. Two former German scientists, Dr. Hermann J. Schaefer and Dr. Dietrich E. Beischer, joined the Research Department in 1947. Research in disorientation acceleration and vestibular functions continued. "Operation Everest," a controlled ascent in the low pressure chamber while acclimatization was studied, was one of the projects of this period.

Peacetime operations prevailed in the Fleet. This situation was faced with a rude shock with the Berlin crisis, resulting in the massive airlift. It began to appear that this was not true peacetime. On 25 June 1950, the cold war was shattered by the invasion of South Korea by Communists of North Korea. President Truman, on 27 June 1950, ordered U.S. forces to the support of South Korea. On that same day, naval aircraft moved out in patrol actions; and, on 3 July 1950, Air Group 5 from the USS *Valley Forge* made the first air strikes.

A general callup of pilots and remobilization of aviation units resulted from U.S. participation with the United Nations involvement in this "police action." Carrier air forces flew sorties from both sides of the peninsula, and helicopters were used for air-sea rescue.

The School of Aviation Medicine again expanded its training program. From classes of six or seven medical officers, they again grew to 25 and over. In addition, refresher courses of two weeks duration were given to former Flight Surgeons being recalled to active duty. Flight Surgeons again proved their value to the operational and support units at sea and with the Marines ashore during this conflict. One Flight Surgeon lost his life and others were wounded in action. A new generation of Flight Surgeons gained operational combat experience in this conflict.

Another result of Korea was the awakening of the U.S. Government and her people to the need for continued alertness. This was reflected in advancement in naval aviation and in aviation medicine to keep apace. This time there was no demobilization or letdown; the watchword was "readiness."

In July 1951, the School of Aviation Medicine was commissioned as a separate command with a medical officer as Commanding Officer. The first Commanding Officer was CAPT Leon D. Carson. In this period, research continued in acceleration, disorientation, acoustics, and psychology. The first repeat examinations of the "1,000 Aviator" study were accomplished. Clinical observations and research from the Neuropsychiatry Department were being published.

In the early 1950s the Naval Flight Surgeon insignia was redesigned to conform more to the design of the Naval Aviator wings. The winged portion of the aviator insignia was used with the center device being the gold leaf with superimposed silver acorn of the Navy Medical Corps.

In 1955, after several years of work by the Aeromedical Association, the Committee on Medical Education of the A.M.A., and the Board of Preventive Medicine, a program was adopted by the Board of Preventive Medicine which would lead to certification in the new specialty of Aviation Medicine. Many senior Navy Flight Surgeons were able to qualify without examination. Others were qualified after examination. A formal program for advanced training was established at the School of Aviation Medicine in 1955. In September 1955, LCDR David P. Morris became the first

Navy Flight Surgeon to enter this residency program. He completed his training in Aviation Medicine at the School in 1958.

In 1956, a permanent board of medical officers was appointed at the School of Aviation Medicine "to provide prompt and highly competent professional review of the physical qualifications of aviation trainees and to expedite processing of those not qualified to continue training." The senior member of the board was the Commanding Officer of the School of Aviation Medicine.

On 30 April 1957, the Naval Aviation Medical Center was established as a command at Pensacola. The School of Aviation Medicine became a component of that command. The Naval Hospital at Pensacola also became part of the Center. The first Commanding Officer of the Naval Aviation Medical Center was CAPT Lester McDonald. At that time, CAPT Julius C. Early, Jr., was the Commanding Officer of the School, and in July 1957 he was succeeded by CAPT Langdon Newman. CAPT J. V. Land, also a Flight Surgeon, was Commanding Officer of the Naval Hospital.

Continuing advances in aviation and technology were leading toward the exploration of space. Rockets were fired into space with instruments and cameras. Scientists at the School of Aviation Medicine were engaged in research in these areas. On 4 October 1957, the U.S.S.R. launched the first earth satellite, heralding the real beginning of the Space Age. In this country, the growth of space technology and research was accelerated. The School of Aviation Medicine entered into research in the problems of space, the selection of candidates to become astronauts, and the development of life support systems.

The first suborbital rocket flight of a primate took place in 1958. A monkey named "Able" was fired into space, survived the journey, but was lost when the nose cone sank at sea before recovery. This experiment was repeated on 28 May 1959, when a squirrel monkey, "Baker" by name, was catapulted into space and recovered alive. Scientists at the School of Aviation Medicine were responsible for the design and fabrication of the biocapsule and instrumentation for both of these flights.

In 1958, a room was built on the hub of the old centrifuge at the School of Aviation Medicine, producing a closed environment that could be rotated. Experiments were begun to determine the effects of rotation on normal and labyrinthine-defect subjects in this "slow-rotating room" over long periods of exposure.

On 4 May 1961, Strato Lab No. 5, a Navy high-altitude balloon flight, made an ascent from the deck of the carrier USS *Antietam*. CDR Malcolm D. Ross was the pilot with LCDR Victor A. Prather, a Naval Flight Surgeon, as medical investigator. Ascent was made to an altitude of over 113,000 feet with a flight duration of 8.9 hours. The two occupants were protected from the effects of reduced barometric pressure by their full pressure suits, since the gondola was of open framework design. This sucessful experimental flight was marred by the death of LCDR Prather by drowning during the recovery phase of the flight.

The continued growth and development of naval aviation and its use in crises and confrontations had kept naval aviation medicine growing and developing. Operational units required Flight Surgeons, and the development of new aircraft and techniques required new aeromedical adjustments, research, and applications.

The space program required support by all services until its transfer to a separate agency of the Government. Since that time, naval aviation medicine research has continued investigation into this field, and Navy avation medical scientists and Flight Surgeons are serving in support of NASA. All recovery operations of Mercury and Gemini astronauts have been by Navy operational units.

To date, six Naval Flight Surgeons have held the office of President of the Aerospace Medical Association. The Navy has supported this association with membership, leadership, and with attendance and presentations at the annual sessions. Of the awards which the Association has presented, naval aviation medical personnel have received recognition under each of the award categories for a total of over 15 individual awards.

On 22 July 1964, a new Coriolis Acceleration Platform and Vestibular Unit was dedicated at the School of Aviation Medicine. This device is a more sophisticated rotating platform to be used to continue the research inaugurated by the old "slow-rotation room." With this great expansion of research and clinical activities, the school occupied many separate buildings on the Naval Air Station. Plans were made in 1961 to build a new school building to bring together most of these activities under one roof. On 14 May 1965, the new building was dedicated. It consists of two structures with 90,000 square feet of floorspace. One structure houses research activities and one is used for training, clinical services, and the Aviation Examining Room.

At present, three classes of medical officers are convened for training in aerospace medicine annually. About 140 Flight Surgeon graduates are produced each year. The academic syllabus lasts 14 weeks after four weeks of indoctrination. The trainee completes his work with a six-week flight indoctrination syllabus leading to the designation of Naval Flight Surgeon. As of 14 December 1967, when Class 116 graduated, a total of 3,410 medical officers had graduated from the School. In addition to Navy medical officers, these included doctors from over 12 foreign nations and doctors from the U.S. Army and U.S. Public Health Service.

Also under the Training Department are the programs for training Medical Service Corps officers as Aviation Psychologists and as Aviation Physiologists. A Research Clerk Program is in effect, consisting of 60-day summer periods for ensigns (MC) who have at least one year of medical school. Courses for enlisted men are given, leading to designation as Aviation Medicine Technician, Aviation Physiology Technician, and Audiometric Technician.

The curriculum for the Aerospace Medicine Residency Program currently is being revised. Plans call for six residents a year to enter the program, providing a total of 18 residents in training at any one time. All residents will begin their advanced training with an academic year at a School of Public Health, leading to the degree of Master of Public Health. This will be followed by two years of advanced training at Pensacola.

The Research Department is carrying out research in aviation and space medicine, experimental psychology, and aviation physiology. Specific areas of study are biophysics, biomagnetics, zero-gravity effects, pulmonary physiology, the 1,000 aviator longitudinal study, vector cardiography, ballistocardiography, exercise ECG, medical statistics, vestibular physiology, medical electronics, physiological optics, biochemistry, neurophysiology, acoustics, audiology, electroencephalography, experimental psychology, aviation psychology, and psychophysiology. To support this research effort, there is a large staff of Navy Medical Corps officers, Medical Service Corps officers, civil service scientists and workers, and enlisted personnel.

The School of Aviation Medicine received a new designation on 2 September 1965. It is now the Naval Aerospace Medical Institute. This change in designation reflects a continuing expansion of the scope of activities of Navy Flight Surgeons as naval aerospace medicine responds to the diverse challenges of the future as it has responded in the past.

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CHAPTER 2

Administrative Control in Operational Aerospace Medicine

Organizational Structure
    Bilinear System of Organization
    Operating Forces of the U.S. Navy
    Administrative Command Within the U.S. Navy
The Navy Directives System
Official Correspondence
References

THE EFFICIENCY with which a Flight Surgeon performs his day-to-day duties will at least in part result from his understanding of the structure and functioning of the U.S. Navy. In particular, a Flight Surgeon must know where he fits into the Navy structure and how appropriate use of this structure will add to his effectiveness. The practice of naval aerospace medicine must be consistent with the organization and procedures of the entire Navy.

ORGANIZATIONAL STRUCTURE

The Department of the Navy is separately organized under the Secretary of the Navy. It operates under the authority, direction, and control of the Secretary of Defense. It is composed of the executive part of the Department of the Navy; the Headquarters, U.S. Marine Corps; the entire operating forces, including naval aviation, of the U.S. Navy and of the U.S. Marine Corps, and the reserve components of those operating forces; and all shore (field) activities, headquarters, forces, bases, installations, activities, and functions under the control or supervision of the Secretary of the Navy. It includes the U.S. Coast Guard when it is operating as a service in the Navy.

The term "Navy Department" refers to the central executive offices and bureaus of the Department of the Navy located at the seat of the Government. The Navy Department includes the offices of all members of the executive administration of the Department of the Navy. These organizationally comprise the Office of the Secretary of the Navy, the Office of the Chief of Naval Operations, and the headquarters organizations of the U.S. Marine Corps, of the Naval Material Support Establishment, of other supporting organizations, and of the U.S. Coast Guard when it is operating as a service in the Navy.

Figure 2–1 shows the basic structure of the Navy Department, attending for the most part to those activities which are of interest to a Flight Surgeon.

The Department of the Navy is organized in four principal parts. These are:

1. *The Operating Forces of the Navy,* which includes the Office of the Chief of Naval Operations, the several fleets, seagoing forces, Seas Frontier Forces, District Forces, Fleet Marine Forces and other assigned Marine Corps forces, the Military Sea Transportation Service, and such Navy shore (field) activities and commands as are assigned by the Secretary of the Navy.
2. *The U.S. Marine Corps,* within the Department of the Navy, which includes Headquarters U.S. Marine Corps, the Operating Forces of the Marine Corps, Marine Corps Supporting Establishments, and the Marine Corps Reserve.
3. *The Naval Material Command,* which includes the Chief of Naval Material and, as subordinate elements, the Naval Air Systems Command, the Naval Ordnance Systems Command, the Naval Ship Systems Command, the Naval Electronic Systems Command, the Naval Supply Systems Command, and the Naval Facilities Engineering Command.
4. *Other Supporting Organizations,* which include the Bureau of Naval Personnel, the Bureau of Medicine and Surgery, the Office of the Comptroller of the Navy, the Office of the Judge Advocate General, the Office of Naval Research, offices of Staff Assistants to the Secretary, and the shore (field) activities as assigned by the Secretary of the Navy.

Bureau of Medicine and Surgery.—Of particular importance for a Flight Surgeon within the Navy Department is the Bureau of Medicine and Surgery, which is responsible for all medical and allied science programs. This bureau is assigned management responsibility for the organization, financing, operations, and maintenance of both medical and dental activities. The

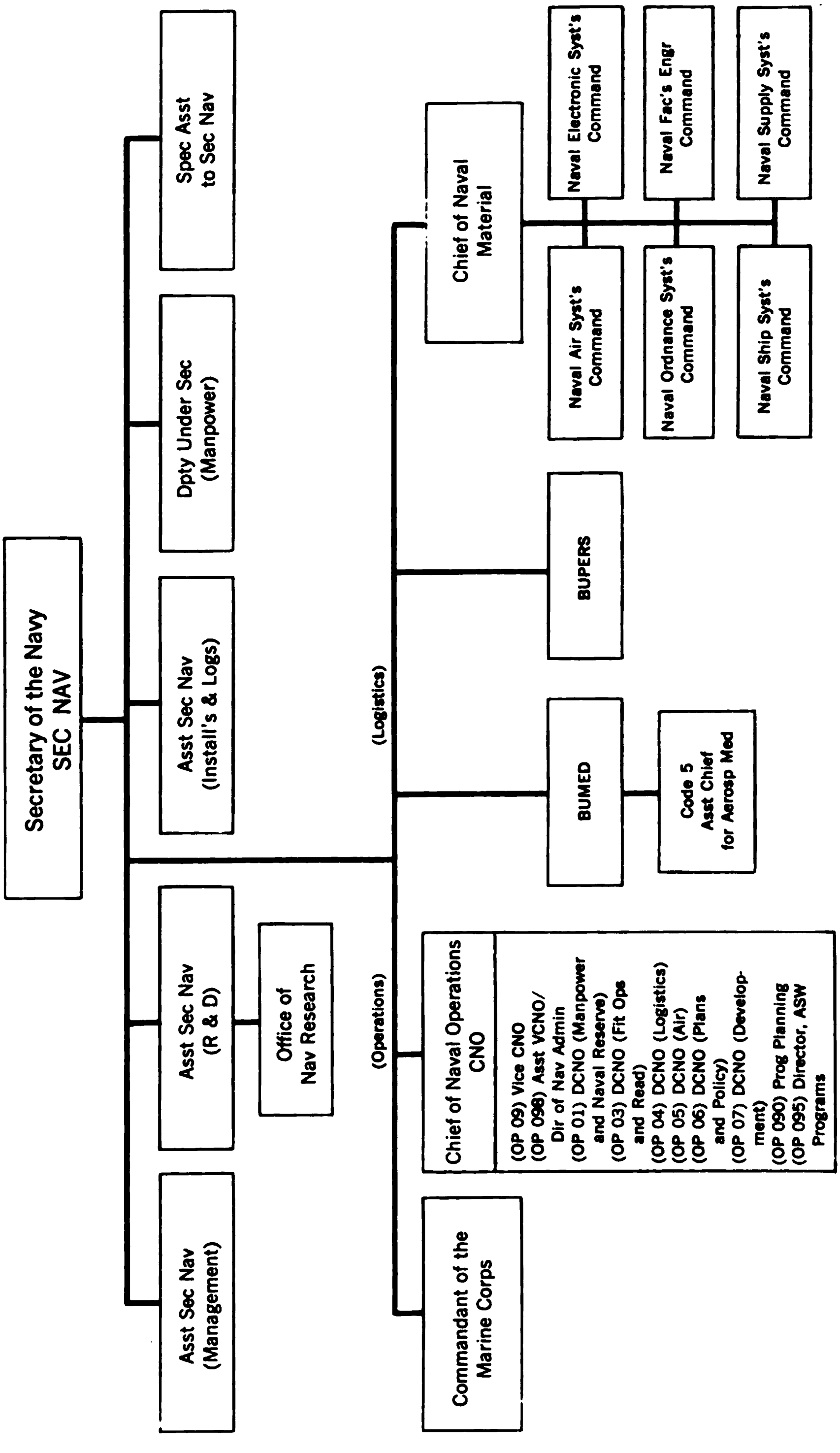

Secretary of the Navy
SEC NAV
Asst Sec Nav (Management)
Asst Sec Nav (R & D)
Office of Nav Research
Asst Sec Nav (Install's & Logs)
Dpty Under Sec (Manpower)
Spec Asst to Sec Nav
(Operations)
(Logistics)
Commandant of the Marine Corps
Chief of Naval Operations CNO
(OP 09) Vice CNO
(OP 09B) Asst VCNO/ Dir of Nav Admin
(OP 01) DCNO (Manpower and Naval Reserve)
(OP 03) DCNO (Flt Ops and Read)
(OP 04) DCNO (Logistics)
(OP 05) DCNO (Air)
(OP 06) DCNO (Plans and Policy)
(OP 07) DCNO (Development)
(OP 090) Prog Planning
(OP 095) Director, ASW Programs
BUMED
Code 5 Asst Chief for Aerosp Med
BUPERS
Chief of Naval Material
Naval Air Syst's Command
Naval Ordnance Syst's Command
Naval Ship Syst's Command
Naval Electronic Syst's Command
Naval Fac's Engr Command
Naval Supply Syst's Command

FIGURE 2–1. *Organization of the Navy Department.*

Bureau also establishes professional standards for clinical methods and procedures in medicine, dentistry, and nursing care and treatment, as well as initiating and directing research in these fields. Finally, in collaboration with other bureaus concerned, the Bureau provides standards for the selection of aviation personnel and for the conduct of specialized sanitation and industrial hygiene practices. The Chief of the Bureau of Medicine and Surgery is The Surgeon General, who is responsible for all medical matters within the Navy.

Within the Bureau of Medicine and Surgery is the Assistant Chief for Aerospace Medicine, who is responsible for all phases of Medical Department programs relating to aerospace medicine and who directs those aerospace medical aspects relating to selection, training, and qualifications of naval personnel for aerospace duties. He also is concerned with medical aspects in the design of aerospace systems and equipment. Figure 2-2 shows those offices within the Bureau directly administered by the Assistant Chief for Aerospace Medicine.

The Aerospace Medicine Operations Division studies, evaluates, and advises on naval and Marine Corps aviation medical needs, policies, standards, practices, and procedures and administers aviation medicine programs relating to physical qualification, training, selection, and aeromedical personnel management. The Division consists of an Office of the Division Director, an Aerospace Physical Qualifications Branch, an Aerospace Physiology Training Branch, an Aerospace Operational Psychology Branch, and an Aerospace Medicine Special Activities Branch.

The Aerospace Medicine Technical Division studies, evaluates, advises, and provides technical biomedical and bioengineering data on those aspects of Navy and Marine Corps aerospace medical programs pertaining to weapons systems requirements, aircraft and space vehicle crews and equipment, aerospace ground support, and aerospace safety. Liaison with the Office of the Chief of Naval Operations and the Naval Air Systems Command is maintained. The Division consists of an Office of the Division Director, an Aerospace Medicine Systems Requirements Branch, an Aerospace Medicine Equipment Branch, and an Aerospace Medicine Flight Safety Branch.

Bilinear System of Organization

The Department of the Navy is unique among the military departments in the bilinear system of organization which it uses. In this system, all

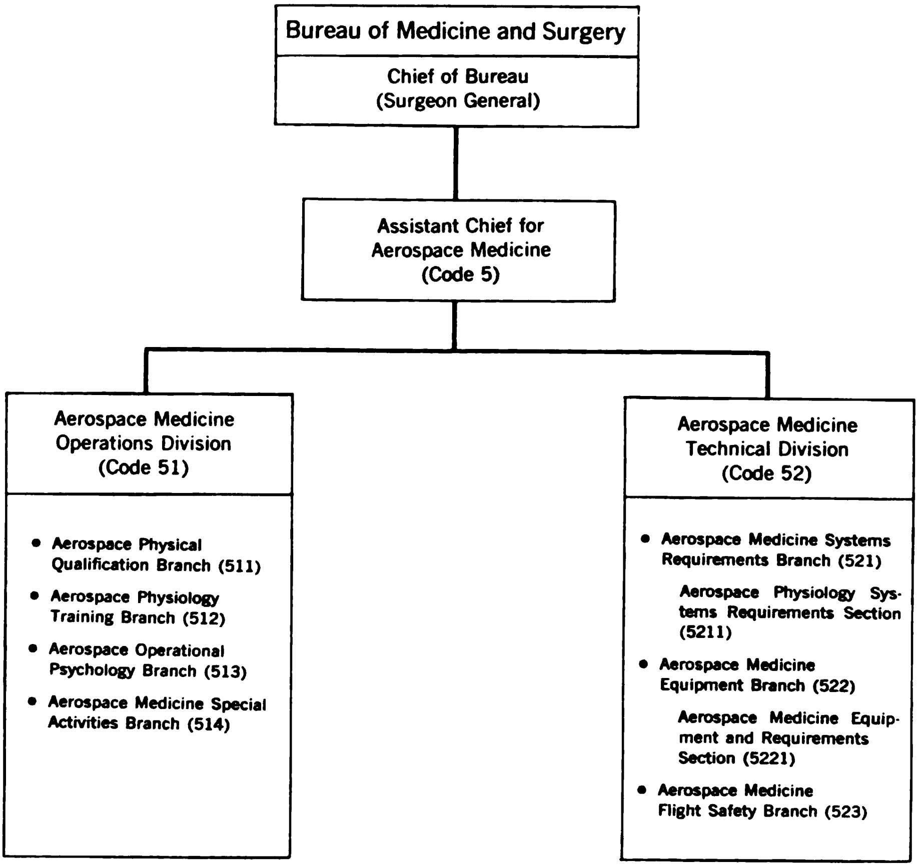

Bureau of Medicine and Surgery
Chief of Bureau (Surgeon General)
Assistant Chief for Aerospace Medicine (Code 5)
Aerospace Medicine Operations Division (Code 51)
• Aerospace Physical Qualification Branch (511)
• Aerospace Physiology Training Branch (512)
• Aerospace Operational Psychology Branch (513)
• Aerospace Medicine Special Activities Branch (514)
Aerospace Medicine Technical Division (Code 52)
• Aerospace Medicine Systems Requirements Branch (521)
Aerospace Physiology Systems Requirements Section (5211)
• Aerospace Medicine Equipment Branch (522)
Aerospace Medicine Equipment and Requirements Section (5221)
• Aerospace Medicine Flight Safety Branch (523)

FIGURE 2-2. *Organization of aerospace medicine activities within the Bureau of Medicine and Surgery.*

major activities, with the exception of the executive offices, can be classified either as "operating forces" or as "administrative or support forces." This bilinear system recognizes the distinction between military direction of the Navy, which is concerned with maintaining appropriate military posture and with the successful accomplishment of assigned missions, and the business direction, which is concerned with providing the personnel, equipment, and services required in support of the military forces.

The bilinear system of organization causes certain overlap in establishing a chain of command for a given individual. This is particularly true in the case of a staff officer such as a Flight Surgeon, who may be serving as part of the operating forces but who may also be under the direct cognizance of a member of the administrative forces. The following sections describe in more detail the command structure of the two parts of the bilinear system.

Operating Forces of the U.S. Navy

The Chief of Naval Operations (CNO) is the senior military officer of the Department of the Navy. He is a member of the Joint Chiefs of Staff and serves as the principal naval advisor to the President and to the Secretary of the Navy on the conduct of war and on the conduct of the activities of the Department of the Navy. CNO coordinates and integrates the requirements of the Operating Forces of the Navy, of the Marine Corps, and of the bureaus and offices of the Navy Department into total requirements for the approval of the Secretary of the Navy. Orders from CNO to operating units in the Fleet are transmitted as shown in figure 2–3 using, for purposes of illustration, the organization of the Pacific Fleet.

The Commander in Chief, Pacific (CINCPAC), receives orders directly from CNO and transmits them in turn to Commander in Chief, U.S. Pacific Fleet (CINCPACFLT). CINCPACFLT splits the Fleet into several forces. Subordinate operational commands of interest to a Flight Surgeon include the 1st Fleet, the 7th Fleet, the Antisubmarine Warfare Force, the Fleet Marine Force, and the Amphibious Force. The 1st Fleet operates in the Eastern Pacific, including the west coast of the continental United States, Hawaii, and the mid-Pacific. The 1st Fleet is an integrated striking force but spends much effort on training and readiness operations. The 7th Fleet serves as a deterrent and ready force in the Western Pacific. This Fleet includes all operating Navy and Marine Corps forces, as well as their mobile support forces.

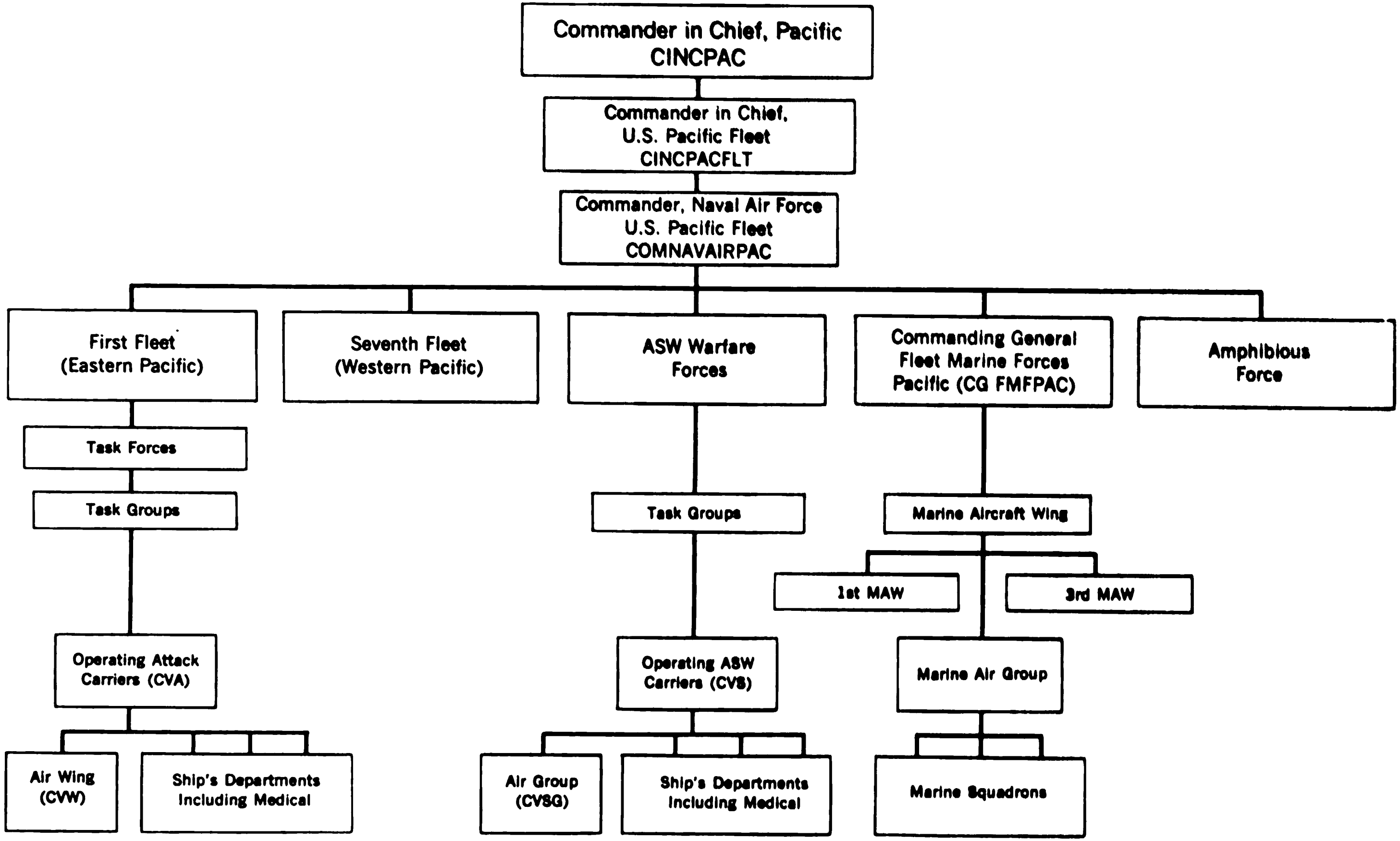


FIGURE 2-3. *Principal operating and logistic force commands within the U.S. Pacific Fleet.*

Carriers and embarked Air Wing/Groups are assigned to task forces for operational control. Task forces may in turn be subdivided into task groups, with one carrier typically assigned to each group.

Operating units aboard an aircraft carrier are under the command of an Air Wing (CVW), in the case of an attack air wing, or an Air Group (CVSG), in the case of an ASW carrier. Aboard ship, the Air Wing/Group Commander serves as a department head and is under the direct control of the Commanding Officer of the ship. Administration and conduct of shipboard activities are handled by a number of departments, each of which reports to the Commanding Officer via the Executive Officer of the ship, just as the CVW or CVSG. These departments include the Executive, Navigation, Operations, Communications, Air, Weapons, Supply, Engineering, Medical, and Dental.

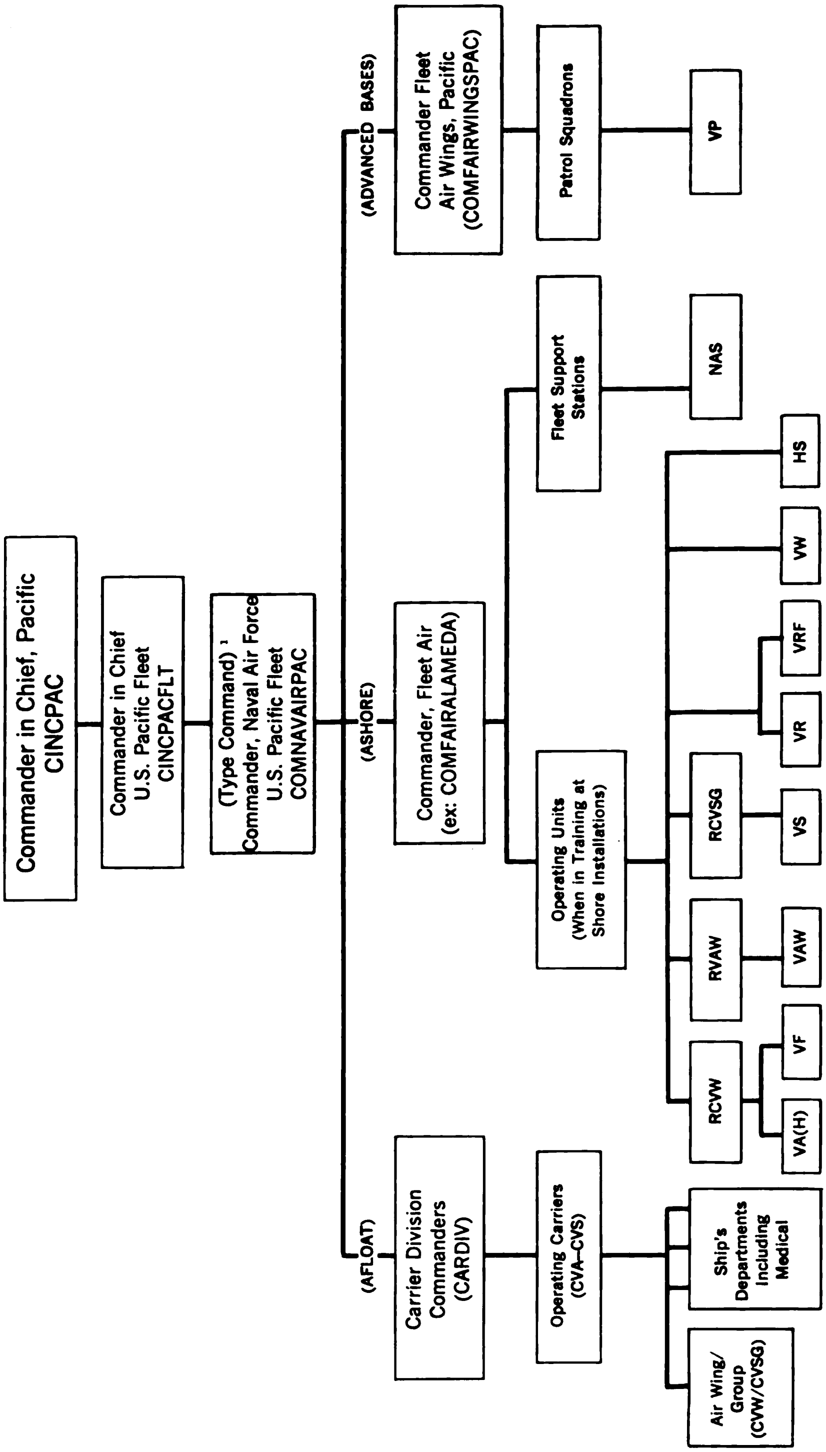
Administrative Command Within the U.S. Navy

Fleet administrative and support forces are controlled, as are the operating forces, through the Fleet Commander. Figure 2–4 illustrates the manner in which this is accomplished, again using the Pacific Fleet structure as an example.

The Commander in Chief, U.S. Pacific Fleet, has under his direct control a number of Type Commands. These Type Commands include Naval Air Forces, Mine Forces, Submarine Forces, Service Forces, Training Commands, Amphibious Forces, Fleet Marine Forces, and Cruiser-Destroyer Forces. Figure 2–4 shows the command structure of a Type Command, the Naval Air Force, in which a Flight Surgeon may expect to serve.

Type Commands exist under the theory that maintenance, supply, manning, and training of tactical units can best be done by types. Thus, for aviation units in the Eastern and Western Pacific areas, all such support comes through Naval Air Force, Pacific. The Commander, Naval Air Force, Pacific Fleet, thus is the producer of the air striking power product which the tactical fleets put to use.

COMNAVAIRPAC staff is divided into five groups covering Personnel, Communications, Supply, Material, and Readiness, plus an additional eight special sections. One of these special sections is headed by the Force Medical Officer, who is in charge of all aviation medicine activities falling within AIRPAC jurisdiction.



¹ Type Commands: Naval Air Forces, Mine Forces, Submarine Forces, Service Forces, Training Commands, Amphibious Forces, Fleet Marine Forces, Cruiser-Destroyer Forces.

FIGURE 2–4. *Principal administrative commands within the U.S. Pacific Fleet.*

Operating carriers under the control of COMNAVAIRPAC are assigned to Carrier Division Commanders. There are two types of CARDIVs: (1) attack carrier divisions, and (2) ASW support carrier divisions. Under CINCPACFLT, there typically are four attack carrier divisions and three ASW support carrier divisions.

Carrier Divisions are administrative commands. Whenever a Carrier Division commander exercises operational command, he does so as a commander of a task group or a task force. For example, the USS *Enterprise* (CVAN–65) at one time was under the administrative command of COMCARDIV 2 and under the operational control of Carrier Task Group 60.1. The Commander, Naval Air Force, Pacific Fleet, also exercises control over a number of Fleet Air Commands and Fleet Air Wings. The Commander, Fleet Air Wings, Pacific, exercises control over all patrol squadrons (VP) in this area. The shore-based Fleet Air Commands serve as geographical representatives of COMNAVAIRPAC and provide training facilities, coordination of maintenance activities, and logistic support for the Operational Forces. Air Wings and Air Groups which are in training in preparation for deployment are under the control of a Commander, Fleet Air Unit. The Fleet Air Command also operates Fleet support stations, such as Naval Air Stations.

THE NAVY DIRECTIVES SYSTEM

The efficiency with which information is disseminated throughout the Navy, as in all organizations, bears directly on its effectiveness. There are four basic sources of information for a Flight Surgeon. These are:

General Orders, Numbers 1–21, Department of the Navy.
Manual of the Medical Department, U.S. Navy. NAVMED 117.
Manual of the Bureau of Naval Personnel. NAVPERS 15791A.
Navy Regulations.

The above are basic references concerning Navy policies. These references are elaborated by more specific information provided through the Navy Directives System. It is quite important that every Flight Surgeon understand and be able to work with the Navy Directives System, since this system is an important technique for insuring appropriate communication and control within the Navy command structure. A directive is defined as an instruction or notice that prescribes or establishes policy, organization, conduct, methods, or procedures; requires action or sets forth information essential to the effective administration or operation of activities concerned; or contains authority or information that must be promulgated formally.

All offices and activities within the Navy may issue directives as required. Major activities, such as the Office of the Secretary of the Navy, the Chief of Naval Operations, and the Bureau of Medicine and Surgery, use directives for the dissemination of policy information. Directives also may be issued by smaller activities, as, for example, the medical department of a carrier.

An *Instruction* is a directive containing authority or information having continuing reference value or requiring continuing action. It remains in effect until superseded or otherwise canceled by the originator or higher authority. In the Marine Corps, this is designated an *Order.*

A *Notice* is a directive of a one-time or brief nature with a self-canceling provision that has the same force and effect as an *Instruction.* Usually it will remain in effect for less than six months, but is not permitted to remain in effect for longer than one year. In the Marine Corps, this is designated a *Bulletin.*

Every directive has a designation, such as SECNAVINST 5215.1B. In this designation, the following coding is of importance:

5215—Standard subject classification number obtained from the current edition of SECNAVINST 5201.11, the Navy-Marine Corps Standard Subject Classification System.

.1—Numerical sequence of directives issued by this originating authority within this subject classification, i.e., the number of directives bearing on this specific topic.

B—The number of revisions. For example, B indicates the second revision of this instruction.

The following are the broad subject groups covered by the Navy Directives System:

Series	*Subject Group*
1000–1999	Military Personnel.
2000–2999	Communications.
3000–3999	Operations and Readiness.
4000–4999	Logistics.
5000–5999	General Administration and Management.
6000–6999	Medicine and Dentistry.
7000–7999	Financial Management.
8000–8999	Ordnance Material.
9000–9999	Ship Design and Ships Material.
10000–10999	General Material.
11000–11999	Facilities and Activities Ashore.
12000–12999	Civilian Personnel.

OFFICIAL CORRESPONDENCE

A Flight Surgeon must know how to communicate with higher authority effectively. This allows him to utilize the vast resources of the Navy in the solution of his problems. Previous sections described the command structure within the Navy and the manner in which information, in the form of directives and instructions, is disseminated downward. This section describes the manner in which information is transmitted from a Flight Surgeon to higher authority through the chain of command. The chain of command exists to facilitate lateral and vertical communication and to bring the resources of every level of command to bear on the solution of problems.

The following are the principal types of Navy communications which a Flight Surgeon might use.

1. *Naval Letter.* The naval letter is used by all components of the Department of the Navy as a formal means of military communication. It may also be used for communications to other agencies, both in and out of Government. As a naval letter proceeds through the chain of command, it is endorsed as required by each higher echelon. The endorsement offers an opportunity to the "via" addressee to furnish both facts and opinions to the addressee. Thus, when the original letter, with all endorsements, is received by the addressee, he knows what all his subordinates know and think about the matter under discussion.

As an example, assume that a Flight Surgeon aboard a carrier wishes to contact the Naval Air Systems Command concerning some problem with aviators' protective equipment. He would prepare a letter for the unit commanding officer's signature. The letter would leave as a communication from the Commanding Officer to the Naval Air Systems Command, in the following form:

Typist's Initials ————————————————→
Preparing Officer's Code ——————————————→
Unit of Origin ——————————→ CVA-66/4/def
Ship's Correspondence Serial Number ——————→ Ser: 1234
1 January 1967

From: Commanding Officer, USS *America* (CVA-66)
To: Naval Air Systems Command Headquarters (Code AIR531)
Via: (1) Commander, Naval Air Force, U.S. Atlantic Fleet
(2) Commander in Chief, U.S. Atlantic Fleet
Subj: Aviators' protective equipment, functioning of
Ref: (1) OPNAVINST 0000 of 1 Jan 1965

The letter, as headed above, will go through the appropriate chain of command to the Naval Air Systems Command. The exact chain of command is not of major concern to the Flight Surgeon inasmuch as the administrative officer of the command will see that the appropriate routing is used.

Endorsements.—As a letter proceeds through each level of the chain of command, that command will include an endorsement which concurs, supplements, or disagrees with the basic letter. Frequently, the endorsement will be no more than simply "Forwarded." Each "via" addressee has 30 days in which to prepare the endorsement and forward the letter.

Advance Copies.—If deemed desirable, carbon copies may be sent to the action addressee and any or all "Via" addressees at the time the original is mailed. This gives them notice of the letter's content and is frequently of considerable help. It affords leadtime in assembling data to be used in their endorsement upon receipt of the original letter, which proceeds normally through the chain of command.

Copies for Information.—Any number of carbon copies of a letter may be sent to other commands, organizations, or individuals who might have an interest in the information in the letter.

2. *Naval Speedletter.* A speedletter is a form of correspondence used for an urgent routine communication. It carries the same requirement for urgent handling as the naval message, but is forwarded by mail rather than by electrical transmission.

3. *Naval Message.* Communications of this type are transmitted electrically and therefore may be used at times when mail service is not available. Emergency requests for materials, services, or assistance, as with aeromedical evacuation, could be by naval message, if urgent, or by mail if routine. Messages are handled under a very strict priority system.

REFERENCES

Department of the Navy, Bureau of Naval Personnel and Navy Training Publications Center. *Naval orientation.*

Department of the Navy. *Bureau of Naval Personnel manual.* NAVPERS 15791A. U.S. Government Printing Office.

Department of the Navy. General Order No. 5, Assignment and distribution of authority and responsibility for the administration of the Department of the Navy.

Department of the Navy. *Manual of the medical department, U.S. Navy.* NAVMED 117. U.S. Government Printing Office.

Department of the Navy. The Navy directives system. SECNAVINST 5215 series.

CHAPTER 3

Assignments, Duties and Responsibilities of a Naval Flight Surgeon

- Assignments
- Air Wing Duty While Deployed
  - Responsibilities of the Flight Surgeon
  - Medical Facilities Aboard a Carrier
  - Activities of Flight Surgeon
- Air Wing Duty While Ashore
  - Responsibilities of the Flight Surgeon
  - Medical Facilities at a Naval Air Station
- Duty With Fleet Marine Force
  - Marine Corps Organizational Structure
  - Marine Aviation Medical Service
- Additional Duty Assignments to Courts and Boards
  - Aircraft Accident Board
  - Naval Aviator/Naval Flight Officer Evaluation Boards
  - Special Board of Flight Surgeons
  - Board of Senior Flight Surgeons
- Special Assignments
- References

ASSIGNMENTS

UPON THE completion of his training at the Naval Aerospace Medical Institute, a Flight Surgeon may be assigned to one of a number of operational billets. The following are commands for likely assignment:

1. Air Wings, Groups, or Squadrons, afloat, ashore, or at advanced bases.
2. Naval and Marine Corps Air Stations, auxiliary air stations, and air facilities.
3. Aircraft carriers.
4. Fleet Air Wings.
5. Fleet Marine Force Aircraft Wings, Groups, or Squadrons, at advanced bases or afloat.

6. Advanced aircraft support or maintenance bases.
7. Fleet logistic wings or Marine aircraft transport groups.

Flight Surgeons may also be assigned to the staff of major aviation commands (such as AIRPAC) or to administrative positions in aerospace medicine as medical liaison officers. After some operational experience, they may apply for residency training in the specialty of aerospace medicine, consisting of an academic year leading to an M.P.H. degree at an approved school of public health, followed by an integrated two-year residency program at NAMI. Flight Surgeons with special training capability, and interest may be assigned duty in aeromedical research and development at various laboratories and operational activities.

Prior to the graduation of a class of Flight Surgeons at Pensacola, a list of billets is sent to the Commanding Officer of the Naval Aerospace Medical Institute. Upon the recommendation of the faculty, he assigns graduates to these billets. In practice, a graduating member is allowed to exercise considerable choice in this matter, but a great deal of weight is given to the academic standing of the individual in his class. Upon completion of the assignment process, the list is transmitted to the Aerospace Medicine Operations Division at BUMED. These assignments are nominated to the Chief of the Bureau of Naval Personnel where the orders actually are issued.

Of the total list of possibilities, it is most likely that a Flight Surgeon will be assigned, for his first tour, to an Air Wing or Group, to the Medical Department of a Naval Air Station, or to a Fleet Marine Force activity. The remaining sections of this chapter describe these assignments in some detail. Assignments other than these three will differ little in terms of the responsibilities of the Flight Surgeon and the medical matters with which he must deal.

In each of the following sections, the discussion is directed primarily toward the activities of a junior Flight Surgeon. Activities of senior Flight Surgeons differ primarily in terms of increased responsibility and additional administrative requirements. Where the role of the senior Flight Surgeon differs, this is indicated.

AIR WING DUTY WHILE DEPLOYED

As a first-tour assignment, many Flight Surgeons are ordered to duty with an Air Wing or Air Group. Under the Level Readiness program for aviation units, an Air Wing[1] always is assembled and trained to an appropriate level of operational readiness. New personnel, including Flight Surgeons, may be assigned at any point during a tour. Thus, a Flight Surgeon might join the unit while it is ashore awaiting deployment or while it is aboard a carrier. This section describes aviation medicine as practiced in an operating Air Wing.

Responsibilities of the Flight Surgeon

A junior Flight Surgeon, when embarked, will be officially assigned to an Air Wing or to a Marine Corps Squadron. In this position, he will be directly responsible to the Wing Commander or to the Squadron Commanding Officer and will advise him concerning all medical matters. The fitness report of the Flight Surgeon will be prepared by the Line Commander. However, the Flight Surgeon always is under the cognizance of The Medical Officer of the ship and performs such duties as The Medical Officer may direct.

The Senior Medical Officer of the ship, whose official title is *The Medical Officer,* also is a Flight Surgeon, typically having the rank of Commander or Lieutenant Commander. The Medical Officer serves as head of the Medical Department and reports directly to the Commanding Officer of the ship via the Executive Officer. The Medical Officer is responsible for maintaining the health of the crew and for advising the Commanding Officer with respect to hygiene and sanitation conditions of the ship.

Medical Facilities Aboard a Carrier

The Medical Department of an aircraft carrier is well-manned, well-equipped, and capable of dealing with virtually any medical situation which might arise among the crew. The size of this department can be appreciated when it is realized that on larger carriers the allowance of the ship is in the order of 5,000 men. In addition, carriers, by virtue of their excellent facilities, serve as the hospital ships of the task force. Carriers often receive patients from ships in company, both at sea and in port. While in port, the carrier usually is designated as medical guard ship, again because of its excellent facilities.

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[1] For the remainder of the manual, the term "Air Wing" will be used to include "Air Group."

In addition to providing medical care for those persons aboard, a carrier is sufficiently equipped to provide nonmilitary medical care of an emergency or routine nature. Carriers are frequently used in the face of natural disaster and can provide extensive emergency care for civilians following earthquakes, fires, and similar catastrophes. Carriers also are equipped to care for women and children (such as dependents at foreign ports) and can even deal with such matters as emergency deliveries.

Staffing.—The officer allowance of the Medical Department of an attack aircraft carrier of the Forrestal Class normally consists of the following:

1 Medical Officer (Head of Department)
1 Surgeon
1 Medical Administrative Officer

In addition, the Medical Department staff is augmented by the services of Flight Surgeons assigned to the Air Wing and/or Marine Corps Squadrons.

The allowance of Hospital Corpsmen assigned varies with the class of ship but normally will be 16 or more, plus additional corpsmen assigned to the embarked squadrons. Assignment of enlisted personnel is by EPDOLANT/PAC (Enlisted Personnel Distribution Office, Atlantic (Pacific) Fleet) through the Force Medical Officer. The following is a typical enlisted rating structure aboard a carrier:

1	HMC	Chief Hospital Corpsman
2–4	HM1	Hospital Corpsman, First Class
3–5	HM2	Hospital Corpsman, Second Class
3–10	HM3	Hospital Corpsman, Third Class
6–8	HN	Hospitalman
2–3	Strikers	

Within the above breakdown are included the following technical specialties:

Specialist	*Enlisted Personnel Classification Code*
Operating Room Technician	HM–8483
Clinical Laboratory Technician	HM–8417
Aviation Medicine Technician	HM–8406
Preventive Medicine Technician	HM–8432
Medical Administrative Technician	HM–8442
Pharmacy Technician	HM–8482
X-Ray Technician	HM–8452

Physical Facilities.—Located in a rather compact area on a carrier, as shown in figure 3–1, are most of the major facilities to be found in a shore hospital, including administrative offices, examining rooms, hospital wards

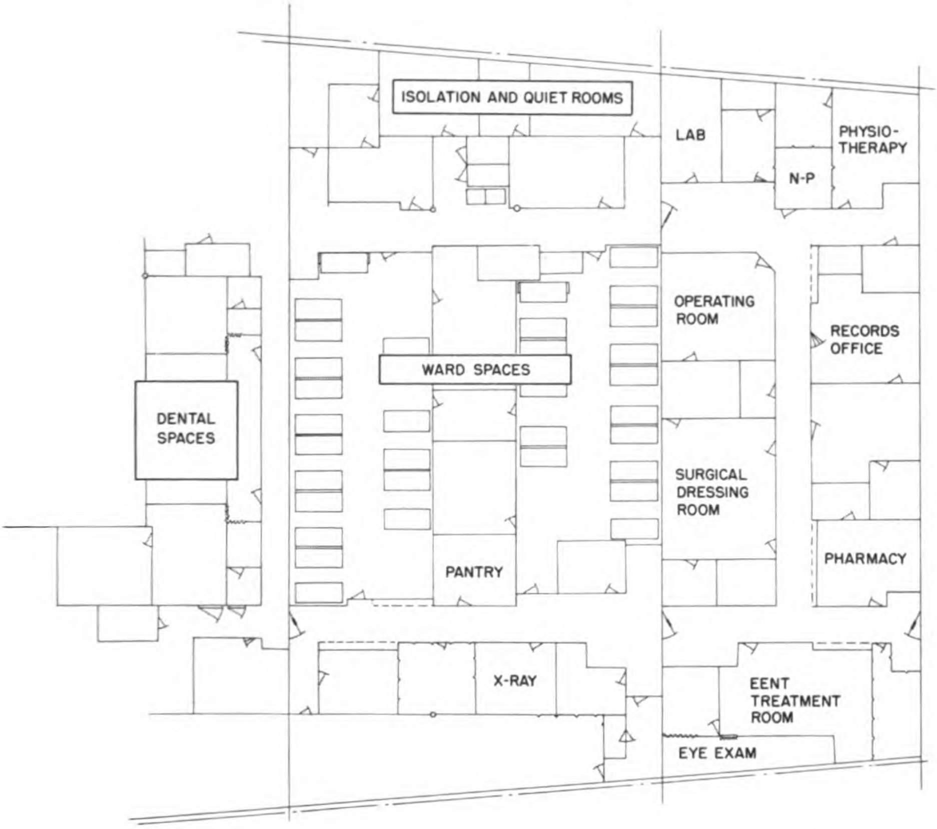

FIGURE 3–1. *Location of medical facilities aboard USS* America *(CVA–66).*

accommodating up to 85 beds, isolation ward, operating suite, ENT clinic, pharmacy, X-ray facility, including a 100-milliampere apparatus or better, diet kitchen, and a clinical laboratory capable of all laboratory work except biochemical determinations. This laboratory is equipped for bacteriological and hematological examinations, including blood typing and crossmatching, and urinalyses. Figures 3–2, 3–3, and 3–4 show the ward spaces, the main operating room, and the ENT clinic, respectively, of an attack carrier.

A carrier also is well-equipped for immediate treatment of injuries received at any point on the ship whether these injuries occur to single individuals during normal operations or to much larger numbers as a result of combat actions. Battle Dressing Stations are situated at strategic locations throughout the ship, as shown in figure 3–5. These stations, which are manned by doctors, dentists, and Hospital Corpsmen during General Quarters, serve as evacuation points for casualties before they are sent on to Sick Bay. The stations are well-stocked with medical instruments, plasma fluids, and other materials required during emergency care. Figure 3–6 shows the layout and equipment of a Battle Dressing Station.

First aid boxes are distributed throughout a carrier, with approximately 220 carried by an attack carrier. These contain dressings, splints, bandages,

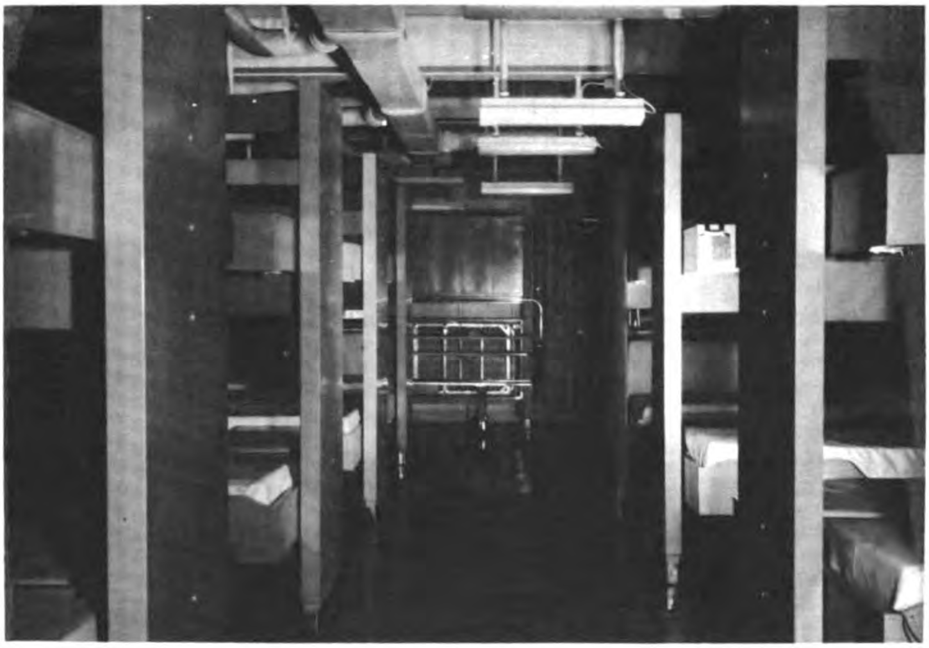

FIGURE 3–2. *View of ward space aboard USS* America *(CVA–66).*

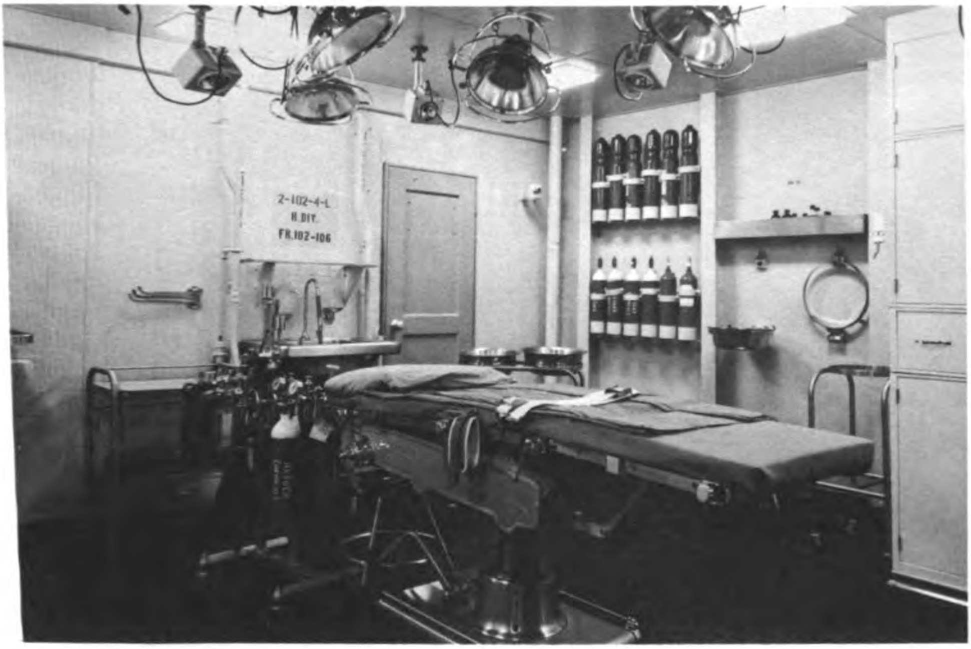

FIGURE 3-3. *Operating suite aboard USS* America *(CVA-66).*

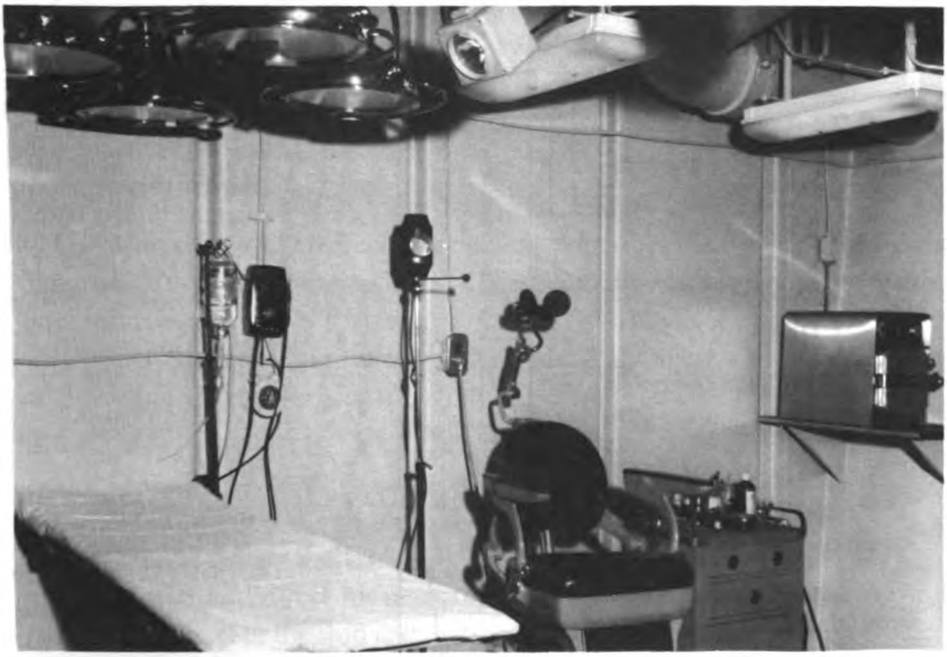

FIGURE 3-4. *ENT clinic aboard USS* America *(CVA-66).*

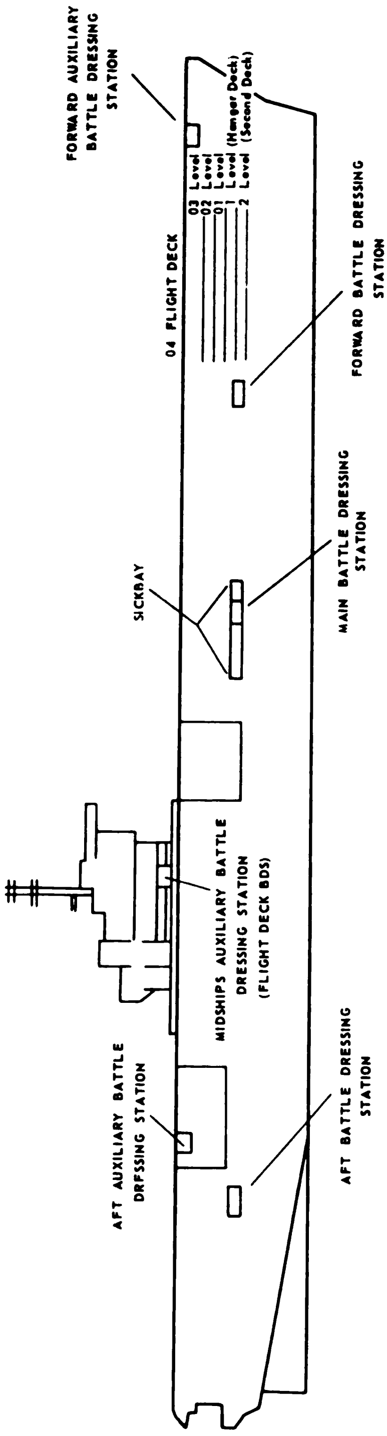
FORWARD AUXILIARY BATTLE DRESSING STATION
04 FLIGHT DECK
03 Level
02 Level
01 Level
1 Level (Hanger Deck)
2 Level (Second Deck)
FORWARD BATTLE DRESSING STATION
SICKBAY
MAIN BATTLE DRESSING STATION
MIDSHIPS AUXILIARY BATTLE DRESSING STATION (FLIGHT DECK BDS)
AFT AUXILIARY BATTLE DRFSSING STATION
AFT BATTLE DRESSING STATION

FIGURE 3-5. *Location of battle dressing stations and sickbay aboard an attack carrier.*

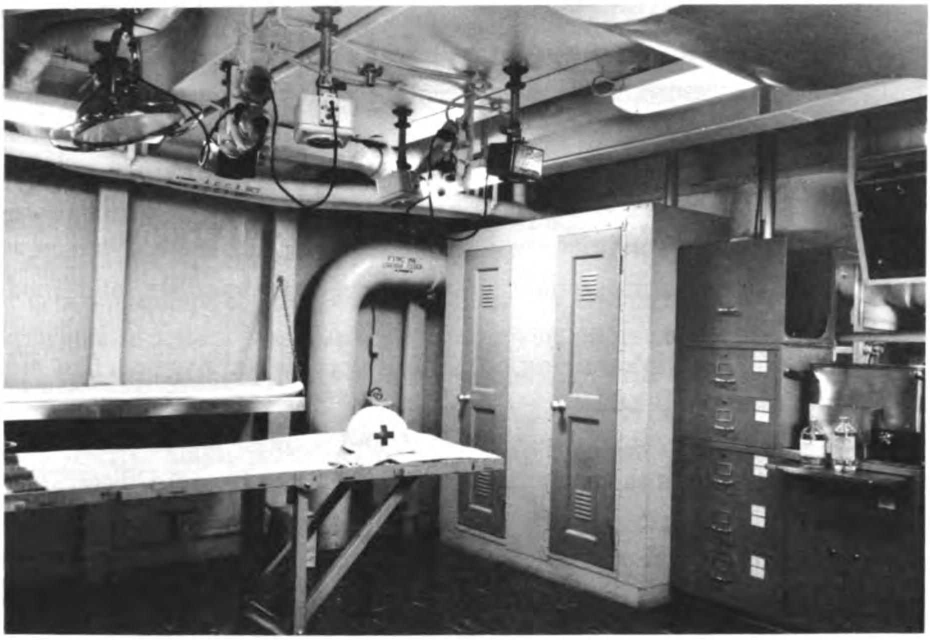

FIGURE 3-6. *View of typical battle dressing station.*

and drugs. There also are about 90 metal stretchers and 30 Neil-Robertson semirigid litters at various locations throughout the ship for use in casualty evacuation.

Activities of Flight Surgeon

The *Manual of the Medical Department* prescribes the functions of Flight Surgeons assigned to aviation units. These functions are presented in guideline form in AIRLANT/AIRPAC CV SHIP INST of the 5400.1 series, which lists the following duties for The Medical Officer and for Air Wing or Squadron Flight Surgeons:

The Medical Officer

The Medical Officer shall:

1. Supervise and direct his department in providing medical and surgical care for personnel of the ship and, when directed by the Commanding Officer, to other personnel in accordance with the *Manual of the Medical Department.*

2. Formulate the policies and procedures of the Medical Department in conformance with the directives of the Bureau of Medicine and Surgery and institute such measures as he may deem necessary to prevent or control disease.

3. Make periodic inspections of the ship's living and working spaces to insure that sanitary measures are being enforced and report to the Commanding Officer any conditions which may endanger the health of the crew.

4. Conduct regular inspections of messmen, food handlers, and food-handling spaces.

5. Supervise the ship's training programs in health and hygiene, first aid, and aeromedical equipment.

6. Procure, inspect, store, issue, transfer, and account for medical stores and equipment except those specifically assigned to the Dental Officer.

7. Refer to the Dental Officer any case discovered requiring dental care and collaborate with him in all cases requiring joint medical and dental care.

8. Advise the Commanding Officer of local quarantine regulations as they affect the ship and assist local health authorities in their inspections.

9. Conduct physical examinations of ship's personnel.

10. Conduct aviation physical and psychological examinations as necessary to determine the fitness of flight personnel for flight status.

11. Recommend to the Commanding Officer the grounding of flight personnel found not physically qualified and/or aeronautically adapted for duty involving flying.

12. Identify and care for the dead.

13. Assume such other responsibilities as may be assigned.

Air Wing Flight Surgeon

The Air Wing or Squadron Flight Surgeon shall:

1. Be responsible, under The Medical Officer, for the medical care and treatment of Air Group and Squadron personnel and assist in the treatment of other personnel when directed by The Medical Officer.

2. Act as consultant to The Medical Officer in all matters pertaining to aviation medicine.

3. Determine by close observation and appropriate clinical investigation the physical and psychological fitness of flight personnel individually and

collectively and initiate appropriate action as dictated by considerations of safety.

4. Conduct examinations for the selection and requalification of flight personnel as directed by The Medical Officer to insure the fitness of personnel for flight duties.

5. Recommend to the Commanding Officer, via the Medical Officer, measures that will contribute to flight safety.

6. Recommend to The Medical Officer suspension from flying, or other appropriate action, whenever in his judgment pilots or aircrewmen are not fit to undertake flight duties without serious risk to the success of the mission or to themselves.

7. Instruct flight personnel in preventive measures regarding conditions peculiar to geographic locations of operations and the medical aspects of aviation safety, as directed by The Medical Officer.

8. Frequently consult with The Medical Officer and Air Group and Squadron commanders and keep them informed on all matters relative to the health and welfare of air group or squadron personnel, particularly those in actual control of aircraft.

9. Supervise the medical personnel of embarked squadrons under the direction of The Medical Officer of the ship and the Air Wing or Squadron Commander.

10. Perform such other duties as may be assigned.

The above are guides for the activities of a Flight Surgeon aboard ship. The following describes in greater detail the manner in which these guides are implemented.

Preventive Medicine.—Aerospace medicine may be considered as a specialized practice within the field of preventive medicine. A very considerable portion of the time of a Flight Surgeon, especially when assigned to an Air Wing or Squadron, is spent in activities related to maintaining the health and flying capability of aviators and aircrewmen. It is his job to see that as many members as possible of the Wing or Squadron remain in flying status. For this reason, a Flight Surgeon is concerned with many matters in addition to the care of the sick or injured.

It is axiomatic that a Flight Surgeon must understand aviation. Toward this end, approximately two months of his training at the Naval Aerospace Medical Institute are devoted to flight training. This qualifies most individuals to solo aircraft. Their education concerning operational aviation should not, however, stop at this point. Every Flight Surgeon should take time to read carefully periodical aviation publications such as *Approach* and *Naval Aviation News.* This will insure that he remains well informed of the many and rapid changes within aviation. ***In addition, every Flight Surgeon should study the NATOPS manuals for the aircraft flown in his unit.*** These manuals present prescribed operating procedures and are particularly useful for their information concerning emergency and ejection procedures.

The *Manual of the Medical Department* states that a Flight Surgeon, in order to discharge his principal duties, shall associate himself with the immediate environment of the pilot as closely as possible. This means that a Flight Surgeon should be familiar with the aircraft and missions being flown by members of his squadron. If at all possible, he should take frequent flights with pilots in his unit. Such flights serve a number of purposes. First, the Flight Surgeon gains valuable insight into the stresses associated with the particular mission being flown. Second, these flights will assist him in assessing aviators and aircrewmen as to their adaptability for the missions they have been assigned. If anxieties and apprehensions are building within a pilot, the Flight Surgeon who participates in such missions on occasion will be more likely to observe the problem and to take corrective action at an appropriate time. Third, these flights tend to establish rapport between the pilot and the Flight Surgeon and, finally, they give the Flight Surgeon excellent insight into the use of aviators' protective clothing and equipment.

It is very important that a Flight Surgeon be familiar with aviators' protective clothing and equipment and the physiological bases for the design of this equipment. There are a number of reasons for this. For one, each item of equipment is designed to protect against a specific environmental stress. The Flight Surgeon should be prepared to explain to aviators the protection which is afforded and the physiological function requiring this protection. Such explanations will do much toward insuring that aviators continue to use the equipment properly. For another reason, a Flight Surgeon can provide those agencies responsible for the development of this equipment with valuable information concerning its adequacy and the manner in which it is being used.

Information concerning proper use and maintenance of every item of protective clothing and equipment is contained in Clothing and Survival Equipment Changes or Bulletins (BACSEBs). These are maintained on file at the Parachute Loft.

While it is of unquestioned importance that a Flight Surgeon understand aircraft, missions, and protective equipment, it is of even greater importance that he associate with and come to know the pilots in his unit. The Flight Surgeon is responsible for monitoring the physical and psychological fitness of flight personnel. In the event an individual appears to be suffering from some physical or psychological condition which might impair his ability to fly, the Flight Surgeon must attempt to correct the condition, using all the resources at his command. If he is successful, only he will be the wiser; if he is unsuccessful, he may have to refer the patient for specialist evaluation and treatment. Certainly, safety of flight must be the Flight Surgeon's paramount consideration. Anything which might be unsafe must be corrected immediately or called to the attention of the Commanding Officer. In addition, in the event of an accident, the Flight Surgeon must describe all personal factors which might have contributed to the accident. This is quite important, since only through the accumulation of a body of information of this type can meaningful conclusions be reached as to the causes which underlie aviation accidents.

In order to meet these responsibilities, a Flight Surgeon should attend ready room briefings and monitor launch and recovery operations. He also may associate with flight personnel during off-duty hours, which must, therefore, make aviation medicine one of the more pleasant of medical specialties.

An excellent opportunity for a Flight Surgeon to maintain close liaison with the pilots in his unit is through attendance at All Pilots Meetings (APMs). These meetings serve to inform him of current unit activities and present a unique opportunity to see all of the pilots of the squadron at the same time.

All of the daily activities of a Flight Surgeon related to monitoring the well-being of Air Wing personnel may be considered as preventive medicine. However, there are a number of very specific preventive medicine tasks, such as food and water inspections, which must be performed by medical personnel of the ship. On an operating carrier, the Wing or Squadron Flight Surgeon will not be involved in these activities to any great extent. However, he should understand the requirements and the mechanics for meeting them. A later section of this manual entitled "Preventive Medicine" discusses these requirements in detail.

Medical Practice.—Although a junior Flight Surgeon aboard ship is assigned to an Air Wing or to a Squadron, he remains an integral part of the Medical Department. The Wing or Squadron Flight Surgeon shares all watches with the other medical officers. He assists the surgeon in any surgery that is performed and also may serve as the anesthesiologist. During flight operations, at least one Flight Surgeon must be immediately available to the flight deck and might at any moment be called upon to administer emergency medical care at the scene of an accident.

During normal operations, much of the time of a Flight Surgeon is devoted to sick call and to physical examinations, including all physicals for flight personnel, as well as, in some instances, safety physicals for flight deck personnel. The Flight Surgeon also is responsible for the immunization of persons within his unit and must see that this is accomplished expeditiously.

Training.—A significant portion of the time of a Flight Surgeon aboard ship is devoted to the preparation and presentation of training materials and lectures. There are a number of communications channels which can be used very effectively by a Flight Surgeon for the dissemination of information. The major channels include:

a. Scheduled lectures as part of an organized training program.
b. Unscheduled lectures on specific problems as they arise.
c. Announcements at Morning Quarters or muster.
d. Ship's intercommunication system—for urgent messages on hazards of a particular area, for example.
e. Closed-circuit television, if available.
f. Ship's Plan of the Day.
g. Bulletin board announcements.
h. Short article or reminder in ship's newspaper.

There are four additional avenues which, although they are not directly a part of the carrier, are very useful. Articles dealing with important topics of broad application in aerospace medicine may be published through:

i. *Approach* Magazine.
j. *U.S. Navy Medical Newsletter.*
k. *Flight Surgeon's News Letter,* published at the Naval Aviation Safety Center, Norfolk, Va.
l. *Naval Aviation News.*

The training effort of a Flight Surgeon covers the following three areas:

1. *Physiological and Safety/Survival Training for Aviators.* The *Manual of the Medical Department* states that Flight Surgeons assigned to operational duties shall participate actively in the Aviation Physiology Training Program

and are responsible for the content of lectures and demonstrations presented to aviation personnel. Such training must cover the topics of physiological aspects of reduced barometric pressure, acceleration, temperature effects, noxious gases, airsickness, disorientation, fatigue, first aid, ionizing radiation, night vision, aviation dentistry, and all the other factors which affect operational readiness. In addition, indoctrination must be given in the use and physiological implications of airborne personal equipment, such as anti-blackout suits, oxygen systems, with particular emphasis on regulators and masks, antiexposure suits, survival equipment, pressure suits, parachutes, protective helmets, noise attenuation devices, ejection seats, escape capsules, and other protective and safety equipment as developed.

All of this training is supplemental to that given at the Aviation Physiology Training Unit ashore and is not intended to supplant it but only to keep it current.

2. *Training of Ship's Crew.* It is the responsibility of the Medical Department aboard a carrier to see that there is an effective, ongoing training program for the crew concerning such topics as emergency first aid, hygiene practices, safety procedures, particularly on the flight deck, and proper use of safety equipment. As a rule, The Medical Officer will contact department heads and request their approval for scheduling of the training to be given. Final schedules are prepared by the appropriate officers in the divisions of the carrier which are concerned.

Closed-circuit television is available for training purposes on some ships. This is an excellent means of presenting material to the crew. With this system, lectures may be taped in advance and shown to a large part of the crew during one session.

The crew training program also should include liberal use of training films, inasmuch as some excellent films are available concerning topics such as emergency care. A complete listing of films can be found in "Film Reference Guide for Medicine and Allied Sciences," published annually by the U.S. Department of Health, Education, and Welfare, and also available as a NAVMED publication (NAVMED 5042).

3. *Medical Department Training.* The Medical Department is responsible for maintaining a continuing program of training for Hospital Corpsmen. This program consists of formal lectures and training films, supplemented by on-the-job training. In the latter program, junior-grade Hospital Corpsmen are rotated through the various services in the department such as the pharmacy, laboratory, and X-ray units. By working with trained personnel,

a corpsman can learn a great deal about all activities within the Medical Department. In case of combat attrition, such knowledge could be critical. Also, and most importantly, such training should assist the corpsman in preparing himself for examinations for promotion.

Flight Surgeons must see that Hospital Corps personnel assigned to aviation activities are thoroughly trained in first aid, with special emphasis on injuries most likely to occur during flight operations. Such training shall include the removal and handling of aircraft casualties, artificial respiration, and the use of resuscitators.

Periodic proficiency checks are made of all Hospital Corpsmen. These checks cover their ability to perform routine tests such as urinalyses and blood counts and to operate Medical Department equipment such as resuscitators, audiometers, and X-ray apparatus. Results of these proficiency tests are entered into the training record of the corpsman. A complete report of all training conducted in the Medical Department is transmitted to the Commanding Officer of the ship through the ship's Training Officer. The Training Office maintains a record of all training accomplished by each department of the ship.

There usually is one or more Hospital Corpsman assigned directly to the Air Wing staff or to an individual squadron rather than to the Medical Department. In these instances, the unit Flight Surgeon is directly responsible for their training, and assignment. Again, however, these matters are subject to final approval by The Medical Officer.

Safety Coordination.—Each ship has a designated Safety Officer, usually a senior commander, and all squadrons have Safety Officers, many of whom are graduates of the intensive course in aviation safety conducted at the University of Southern California. These are the persons with whom the Flight Surgeon must work to insure that a continuous and effective safety campaign is carried out.

Another avenue of approach to a safety program is the use of the Aviation Safety Council. Members of this council include the Commanding Officer of the ship, the Operations Officer, the Safety Officer, the Air Group Commanding Officer, Squadron Commanding Officers and detachment commanders, each Squadron Safety Officer, The Medical Officer, and all Air Wing Flight Surgeons. Agenda items are submitted to the Council by committees, of which the Medical Committee is one, a week in advance. The agenda covers a wide range of items, with flight deck operations, aircraft handling, and flight operations under frequent discussion. A typical agenda item for this council, which meets monthly, might concern the pros and cons of lifejacket use by flight deck personnel. In all, the Aviation Safety Council represents an important avenue for medical personnel to contribute to the overall safety program of a ship or station.

Participation in the Safety Council does not complete a Flight Surgeon's safety responsibilities. In addition, he should monitor the safety practices of individuals in the various aviation units and work with the Safety Officer of each squadron. It is beneficial to check the "Notes from Your Flight Surgeon" in the monthly issues of *Approach*. It will soon be obvious that a great many of these notes deal with improper use of pilot protective equipment and that a continuous effort must be exerted to see that all aviation personnel use such equipment so that it will provide the maximum protective benefit.

Predeployment Preparation.—A Flight Surgeon about to deploy should obtain from the Force Medical Officer a copy of the Cruise Report of the latest carrier to return from the area of concern. This report will provide excellent insights as to conditions likely to be encountered during the cruise. Such reports are prepared by each department of a ship at the conclusion of a cruise and are submitted to the Type Commander.

Prior to embarkation, the Flight Surgeon should make certain that all Air Wing personnel have received all immunization required for the area of deployment, and that all required physiological training has been scheduled by the Aviation Physiology Training Unit. This requires at least a month's leadtime so that the immunization and physiological training records can be checked. This work should be performed by Air Wing corpsmen, and they should be relieved of any duty assignment which might conflict.

Inport duties are shared with other Medical Officers and include consultations and physical examinations for other ships in company. During this period, visits may be made to local medical and health facilities. A visit definitely should be scheduled to the Preventive Medicine Unit, should one be located at the port of call.

AIR WING DUTY WHILE ASHORE

Responsibilities of the Flight Surgeon

A junior Flight Surgeon assigned to an Air Wing or to a Squadron will be directly responsible to the Commanding Officer of that unit. However, when ashore, he may expect to spend much of his time under additional duty orders to the Medical Department. During this period, the professional activities of the Flight Surgeon will be directed by the NAS Medical Officer, although the Air Wing Flight Surgeon remains responsible to the Air Wing Commander for the health and welfare of Air Wing personnel.

While with their aviation units, Flight Surgeons deal with purely aviation medicine matters. These include delivery of lectures on aviation physiology, inspecting safety/survival equipment, monitoring the flight fitness of aviators in the unit, flying with unit aviators, and learning the patois of the unit. Informal or "sidewalk" consultations are held with Air Wing personnel, and the Air Wing corpsmen are monitored in their Air Wing duties. The Flight Surgeon also may attend courses which enhance his professional knowledge.

A junior Flight Surgeon assigned directly to a Naval Air Station Medical Department performs tasks as directed by The Medical Officer. A considerable part of his time is spent in the problems of routine medical care and in conducting aviation physical examinations. All station aviators and aircrewmen, as well as other aviation personnel who might be working at nearby stations having no medical facilities, must be examined periodically.

A number of Naval Air Stations house Naval Air Rework Facilities, which hire a number of civil service personnel. These are large, industrial-type organizations which have special preventive and occupational medicine problems as well as a certain number of emergency conditions. In the event of accident at these facilities, emergency treatment is given at the Station Dispensary.

Each major Naval Air Station has an Aviation Physiology Training Unit. This unit is directed by an Aviation Physiologist who, in turn, reports to The Medical Officer. The Medical Officer is responsible for program syllabi and for the effective operation of this unit. When so assigned, the junior Flight Surgeon is delegated responsibility for reviewing lecture materials, providing medical support during low pressure chamber runs, and monitoring the overall operation of the unit.

Medical Facilities at a Naval Air Station

A certain number of graduates of the Naval Aerospace Medical Institute may expect assignment directly to the Medical Department of a Naval Air Station. They should be familiar with the facilities and activities of the

Medical Department of a Naval Air Station. This department provides medical care for all personnel serving at the Air Station, including Air Wings, plus the dependents of all of these individuals, along with a great many other important services.

Staffing.—The officer allowance of the Medical Department of a Naval Air Station normally consists of the following:

1 Medical Officer (Head of Department)
1 Flight Surgeon
1 Surgeon
3–4 General Medicine Officers
1 Medical Administrative Officer
1–2 Aviation Physiologists
1 Sanitation Officer

In addition, the Medical Department staff may be augmented by the part-time services of Flight Surgeons assigned to Air Wings and/or Marine Corps Squadrons temporarily attached to the Station.

The facilities of certain Naval Air Stations include Station Hospitals. Such hospitals are located at Quonset Point, R.I.; Lemoore, Calif.; Whidbey Island, Wash.; Brunswick, Maine; Twin Cities, Minn.; Lakehurst, N.J.; and Patuxent River, Md., as well as at Atsugi, Japan. The Marine Corps Air Stations at Cherry Point, N.C.; El Toro, Calif.; and Iwakuni, Japan, also have Station Hospitals. The Medical Department staff at these stations may be augmented by six to eight additional Medical Officers. These officers will include specialists in such areas as surgery, obstetrics and gynecology, radiology, pediatrics, and internal medicine.

DUTY WITH FLEET MARINE FORCE

All medical services in support of operations of the Marine Corps are supplied by personnel of the Navy. Thus, graduating Flight Surgeons may expect, as one possibility, assignment to a Marine Aircraft Wing, a Marine Corps Air Station, or a Marine Aircraft Transport Group. In these assignments, the Flight Surgeon may be ashore, afloat, or operating at an advanced base.

As a rule, 17 to 20 percent of all Navy Flight Surgeons are assigned to duty with the Marine Corps. Flight Surgeons ordered to such duty upon completion of study at the Naval Aerospace Medical Institute initially are sent to a two-week Field Medical Service School. This training is conducted at Camp Lejeune on the east coast and Camp Pendleton on the west coast. It consists of basic training in field medicine and orientation to Marine Corps procedures. Training includes familiarization with Marine Corps gear and armament, and instruction in principles of field sanitation. Upon completion of this training, Flight Surgeons are assigned to a Wing or Group or to a Marine Corps Air Station. Recommendations for subsequent squadron assignment are made at the Wing level.

Marine Corps Organizational Structure

Marine Corps operating units are organized within Fleet Marine Forces. As shown in figure 2-3 (of chapter 2), a Commanding General, Fleet Marine Forces, reports directly to an area Fleet Commander.

Marine Aircraft Wing.—The Marine Aircraft Wing, commanded by a major general, is the largest aviation command within the Fleet Marine Force. On the Special Staff is the Wing Medical Officer, whose duties are similar to those of the Division Surgeon. Personnel and materiel requirements are determined at the Wing level for all units, and it is the responsibility of the Wing Medical Officer to insure the highest level of medical combat readiness. In addition, Wing training programs for medical and nonmedical personnel are established, and aviation physiological training for flying personnel is supervised at this level. Assisting the Wing Medical Officer is the Wing Medical Administrative Officer, who is especially concerned with the coordination of administrative functions, medical materiel and logistics, maintenance of records, and the supervision of Hospital Corps personnel. Figure 3-7 presents the essential structure of a Marine Aircraft Wing.

Marine Air Group.—Tactical Marine Air Groups consist primarily of fighter, attack, or helicopter squadrons. As shown in figure 3-7, supporting services such as maintenance and repair, transport and logistics functions, are provided by Marine Aircraft Wing Service Groups, while the Headquarters Group serves the Wing in an administrative capacity. Headquarters Group also contains special units such as Air Control Squadrons, Air Support Squadrons, Light Antiaircraft Missile Battalions, and Photoreconnaissance Squadrons. The number and type of squadrons within the Air Group vary and are not established by any fixed Table of Organization.

Marine Corps Squadrons.—The Tactical Air Groups shown in figure 3-7 are composed of attack, fighter, and helicopter squadrons. The essence of

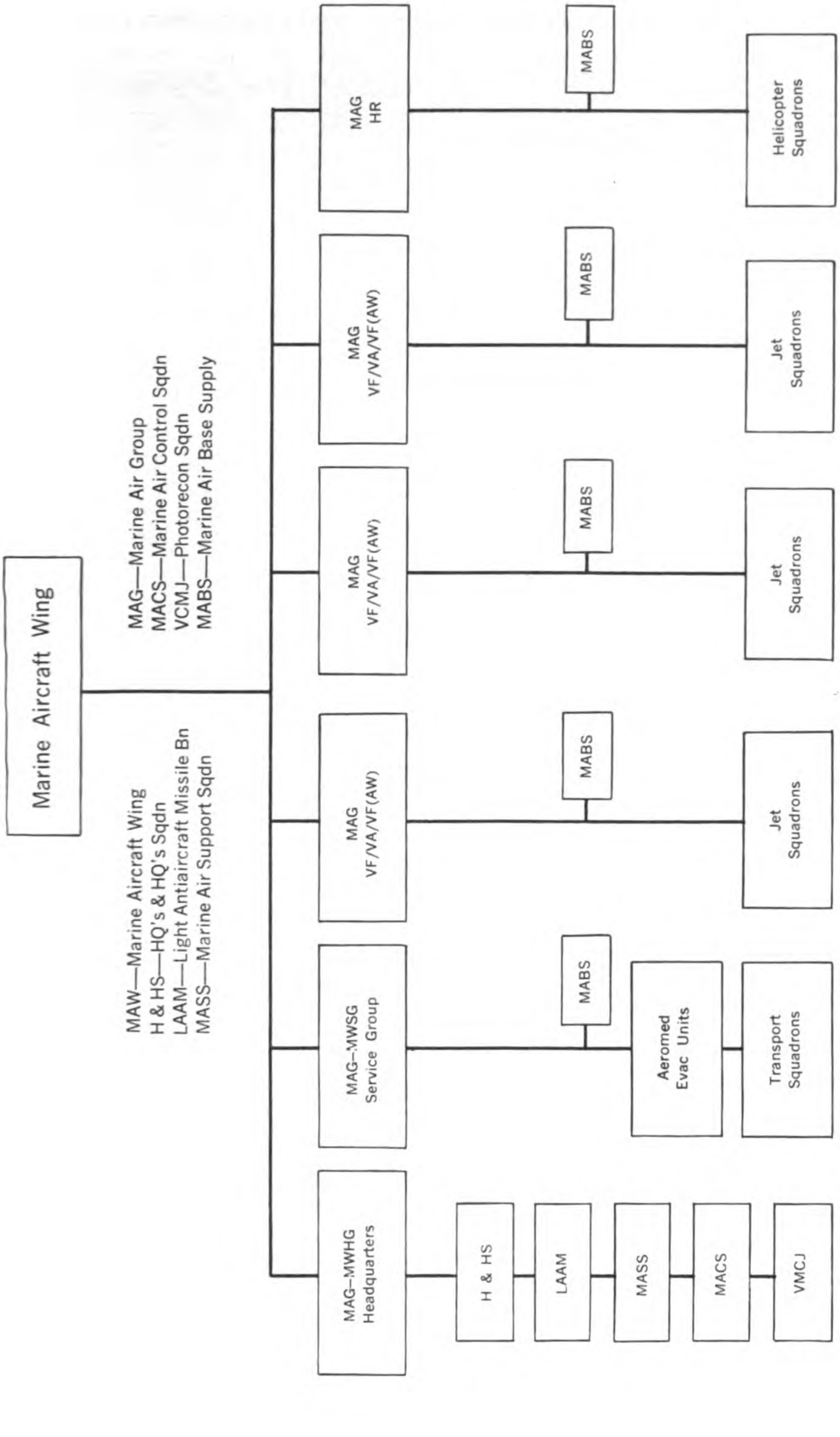

FIGURE 3-7. *Essential structure of Marine Aircraft Wing.*

squadron operation is mobility, with squadrons deploying at times as individual units rather than as a complete Air Group. For this reason, Flight Surgeons are assigned to individual squadrons instead of, as in Navy operations, to Aircraft Wings or Groups with cognizance over one or more squadrons.

Marine Aviation Medical Service

The practice of medicine in Marine Corps units differs somewhat from that within the Navy. Marine units customarily are located at shore-based facilities, with personnel health influenced by climate, terrain, and endemic diseases. Flight Surgeons must understand principles of field sanitation and tropical medicine in order to insure the well-being of their personnel. In addition, if combat conditions prevail, the Flight Surgeon must be prepared to provide emergency treatment for combat casualties, both for members of his aviation unit and, in all likelihood, for personnel of support and ground units operating with the squadron.

A Flight Surgeon assigned to Marine Corps duty has an extra responsibility in that he must be conversant with both Navy medical procedures and those of the Marine Corps. Marine Corps squadrons, on occasion, deploy aboard carriers with a Navy Air Wing. In this event, the Flight Surgeon assigned to the Marine Corps squadron operates exactly like any Navy Air Wing Flight Surgeon.

Marine Field Facilities.—One of the major features of Marine Aviation Medical Units is the capability of independent medical activity for varying periods of time in an operational or combat situation. This is achieved at the Marine Air Group level. Upon deployment, an Air Group carries with it supplies and equipment adequate to establish a 20-bed field hospital. Because of the nature of an Air Group's mission, it is presumed that the hospital will be erected in proximity to an airfield of some kind, whether newly formed or one which has been in prior use. Recent advances in the deployment of specialized aviation equipment have given tactical Marine squadrons the capability of conducting operations from shore airfields in a manner similar to that aboard aircraft carriers.

Figure 3–8 shows the manner in which an Air Group Hospital operates in support of Wing Command activities. The organization shown is that used in the Second Marine Aircraft Wing, with the 20-bed Group Hospital capacity augmented by the 50 additional beds of an Aeromedical Evacuation Unit

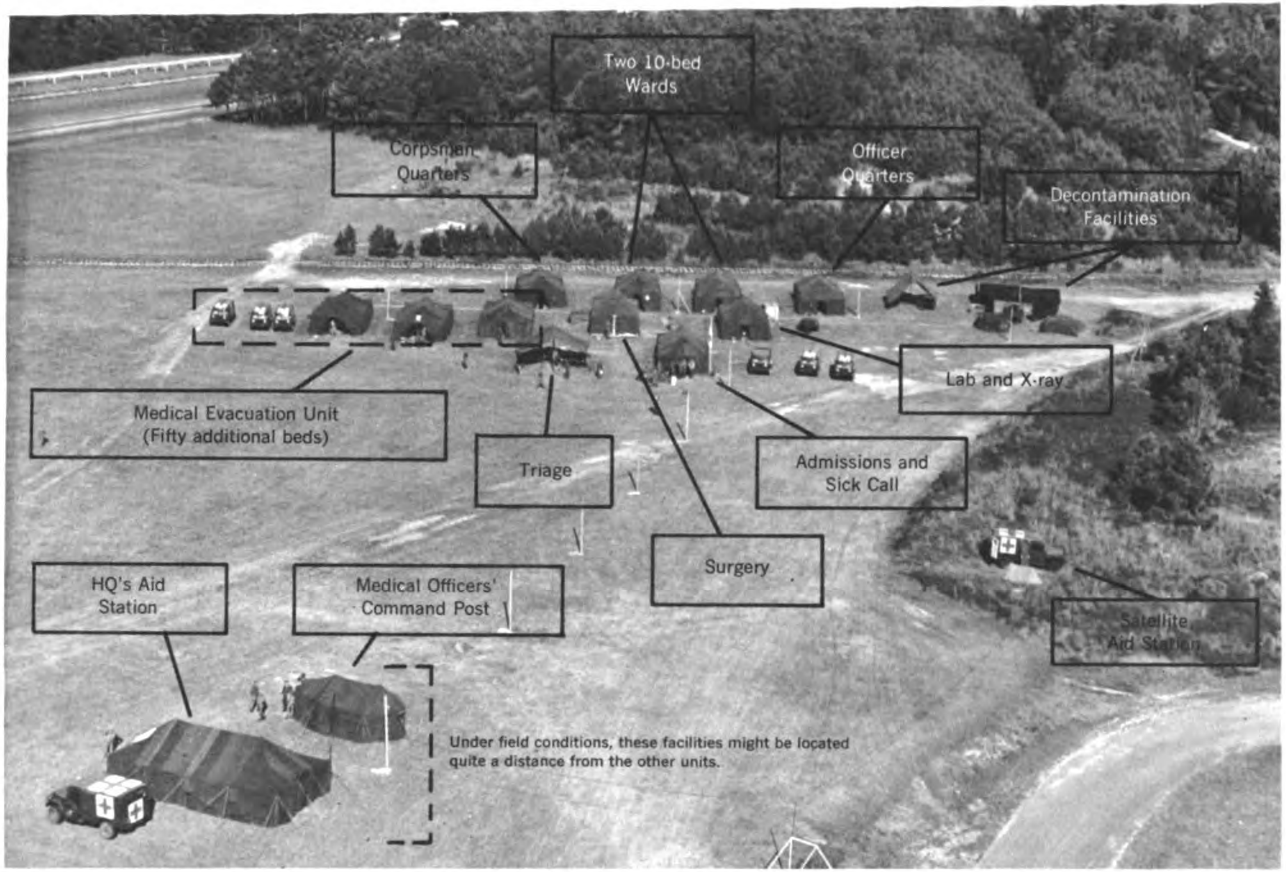
Two 10-bed Wards
Corpsman Quarters
Officer Quarters
Decontamination Facilities
Medical Evacuation Unit (Fifty additional beds)
Triage
Lab and X-ray
Admissions and Sick Call
Surgery
HQ's Aid Station
Medical Officers' Command Post
Satellite Aid Station
Under field conditions, these facilities might be located quite a distance from the other units.

FIGURE 3-8. *Medical facility within a Wing Command Post, showing a Group Hospital augmented by an Aeromedical Evacuation Unit provided by Marine Aircraft Wing Service Group.*

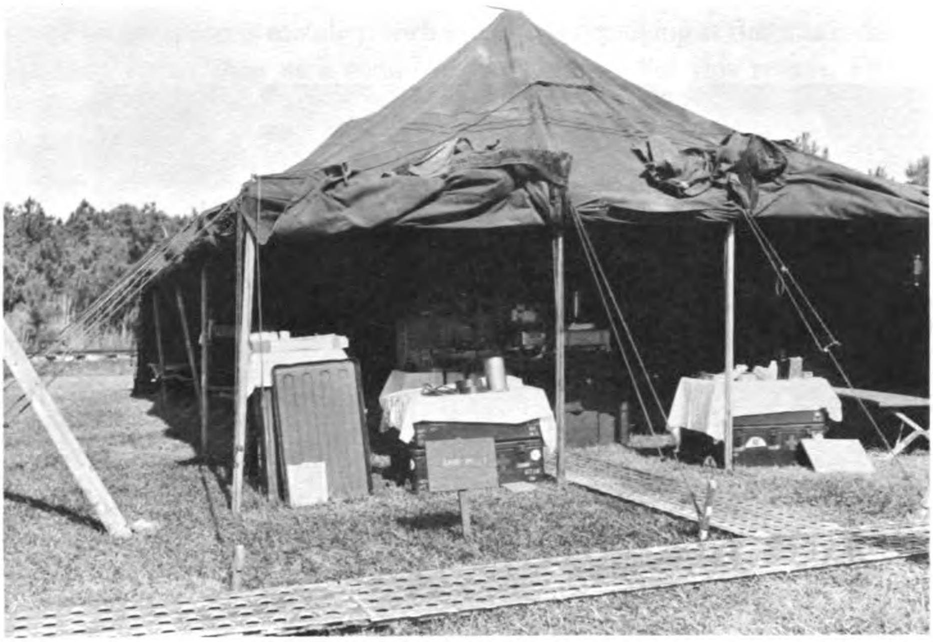

FIGURE 3–9. *Ten-bed field ward, part of Group Hospital.*

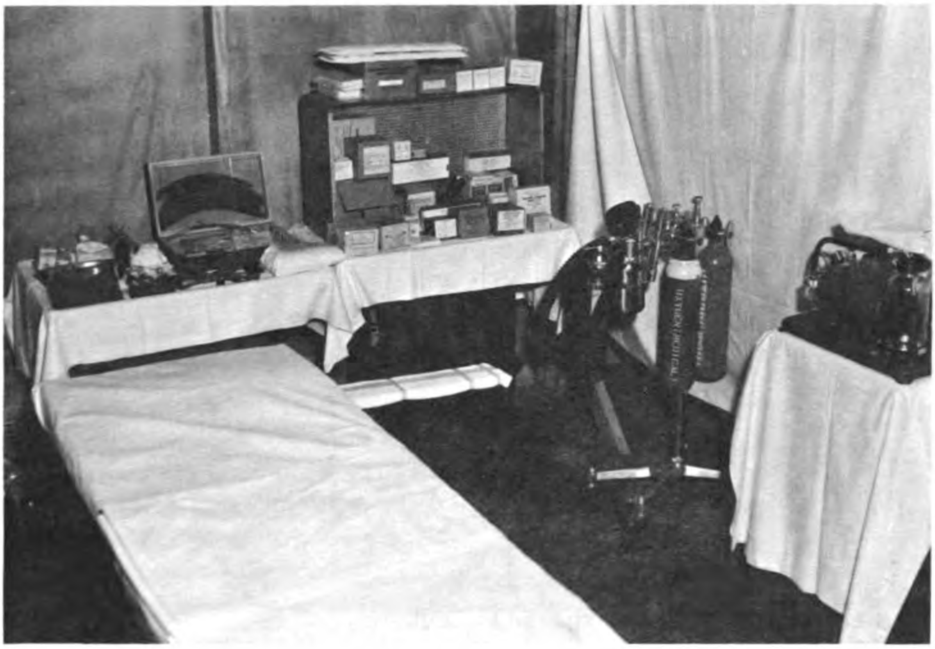

FIGURE 3–10. *Surgical area in field hospital.*

supplied by the Marine Aircraft Wing Service Group. Figure 3–9 shows the 10-bed ward used in a Group Hospital. The associated Field Surgical Facility is shown in figure 3–10. Emergency care of combat casualties can be provided through use of the Satellite Aid Station shown in figure 3–11. This figure illustrates the complete mobility of such units, thereby insuring that medical aid is rendered in as short a period of time as possible following injury.

The increased use of helicopters in aeromedical evacuation means that casualties can be brought to a Group Hospital quite rapidly and that very serious cases, requiring more specialized care, can be transferred without delay to more fully equipped hospital facilities. Figure 3–12 shows the unloading of a casualty from a helicopter.

In the event of actual hostilities, with an Aircraft Wing deployed into a combat area, the personnel within each Group Hospital would be augmented by a complete Surgical Unit, consisting of a general surgeon, an orthopedic surgeon, and an anesthesiologist, and ten specialized hospital corpsmen. This will give the Group Hospital a complete surgical capability.

Training.—All Marine Corps aviators are designated Naval Aviators. Marine Corps aviators receive the same training as Naval Aviators in aviation

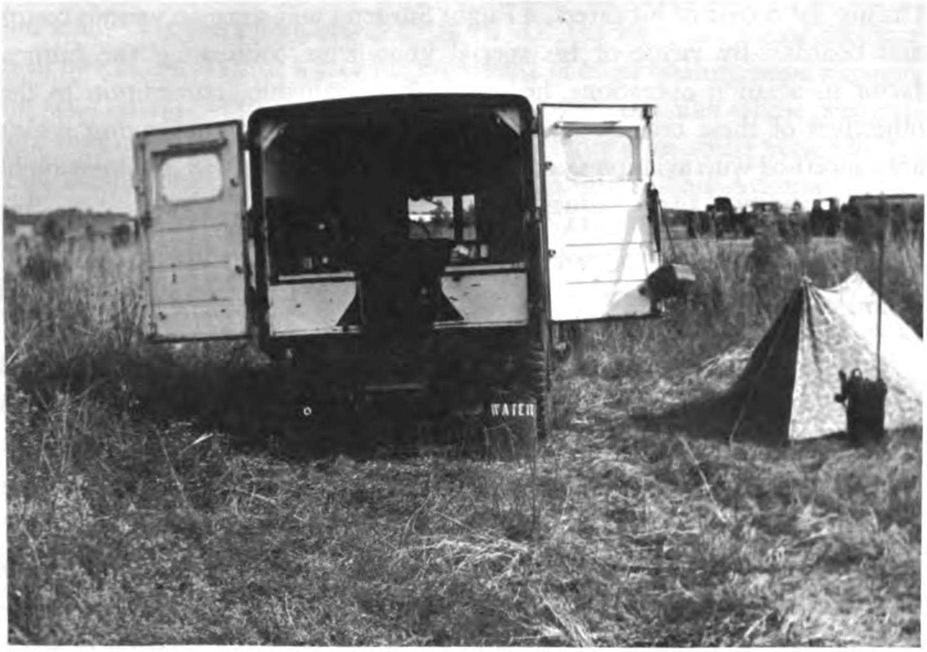

FIGURE 3–11. *Satellite Aid Station.*

physiology, ejection seat techniques, helicopter rescue procedures, and survival and escape methods. A number of Marine Corps Air Stations have Aviation Physiology Training Units operated by Navy personnel. The Marine Corps Flight Surgeon has the same responsibilities with respect to training in aviation physiology as does the Navy Flight Surgeon.

The Second Marine Aircraft Wing recently established an Operational Aviation Medicine and Survival Program. The objective of this program is to present safety and survival information to each aviator, specifically directed to the kind of flying and type of aircraft with which he is engaged. Emergency conditions and aircraft egress procedures pertain specifically to the aircraft being flown. In addition to this safety training, fundamentals in escape and evasion methods, oriented to the specific geographical areas in which a Marine Corps aviator might abandon his aircraft, are included in the program.

ADDITIONAL DUTY ASSIGNMENTS TO COURTS AND BOARDS

During the course of his career, a Flight Surgeon will serve on various courts and boards. By virtue of his special knowledge concerning the human factor in aviation operations, he can make a valuable contribution to the objectives of these boards. Among these boards are the following which are concerned with aviation safety and, in many instances, with the assessment of the capability of an individual to fly.

Aircraft Accident Board

Each Navy aircraft accident, incident, or ground accident is investigated by an Aircraft Accident Board in accordance with OPNAVINST 3750.6 series entitled "Navy Aircraft Accident, Incident, and Ground Accident Reporting Procedures." This board normally is appointed by the reporting custodian for the aircraft involved, but may be one appointed by a higher authority. In addition, in special cases, an independent safety investigation may be conducted by representatives of the Naval Aviation Safety Center. In such case, there is an unrestricted exchange of information between the two investigating efforts.

FIGURE 3-12. *Unloading of casualty from helicopter.*

A full Aircraft Accident Board consists of at least four officers, of whom two must be experienced naval aviators. Of the four, the senior member must be a Naval Aviator with wide experience or other qualifications warranting such an appointment, and he must be senior to the pilot whose performance may come under investigation. One member is an officer versed in aircraft maintenance and engineering. One member is an Aviation Safety Officer, a graduate of the USC Safety Officer Training Course, if available. The fourth member is a Flight Surgeon, or a Medical Officer in the event a Flight Surgeon is not available.

The purpose of an aircraft accident investigation is to determine all contributing causal factors, to reveal facts, and to obtain information which can be used as a basis for corrective action for the prevention of similar mishaps. Reports of these investigations are privileged documents and can be used only for analysis and statistical studies concerned with prevention of future accidents. Results cannot be used as a basis for any legal action or in determining the responsibility of personnel from the standpoint of disciplinary action.

The specialized talents of the Flight Surgeon as a member of the Aircraft Accident Board are directed toward uncovering underlying medical and physical factors which might have contributed to the accident sequence. Thus, such questions as whether fire occurred in flight or after collision with the ground frequently can be determined by the condition of the human remains.

The Flight Surgeon should participate fully in the investigation of the accident and in deliberations of the Aircraft Accident Board. The Flight Surgeon must prepare a Medical Officer's Report (MOR). A later section of this manual discusses in detail the Flight Surgeon's role in accident investigations and the submission of the MOR.

Naval Aviator/Naval Flight Officer Evaluation Boards

Field Naval Aviator/Naval Flight Officer (NFO) Evaluation Boards are convened to evaluate the performance, potential, and motivation of aviation personnel for continued service. The sole purpose of these boards is to support safety of flight operations by determining whether it is in the best interests of an individual or of the Navy for that individual to continue flying. The board does not serve a disciplinary function and must clearly avoid any connotation that its actions are disciplinary in nature.

A Field Naval Aviator/NFO Evaluation Board is convened by the immediate superior in command or the commanding officer of the individual concerned. One member of the board must be a Flight Surgeon. Remaining members are line officers senior to the individual involved.

The following reasons are considered sufficient for convening of this board:

1. The aviator has demonstrated faulty judgment in flight situations. This may be evidenced by serious or repeated violations of flight discipline, serious or repeated accidents involving flight judgment, or repeated minor incidents of any kind in which flight judgment is a factor.

2. The aviator or NFO has demonstrated a lack of general or specific flight skill. This may be evidenced by failure to complete satisfactorily all or any part of prescribed training syllabi or failure to comply with minimum annual flying requirements for reasons within his control. In the case of aviators, this may also be evidenced by accidents or near-accidents involving pilot skills.

3. The aviator or NFO has demonstrated certain habits, traits of character, emotional tendencies, lack of mental aptitude or motivation that make it questionable to continue him in his assigned flying duties.

4. The aviator or NFO is deemed professionally unfit for flying for any reason not specified above.

Aviators and/or NFOs appearing before a field board will be recommended for one of the following classifications:

Type A—Continuation in a flight status.

Type B—Unsuited for continuation in a flight status by reason of lack of general aeronautic ability and/or psychological reasons and/or motivation.

Paragraph C-7318 of the *Bureau of Naval Personnel Manual* lists detailed recommendations considered appropriate under Type A or B classifications. Review procedures and instructions regarding special cases also are presented.

Prior to serving on a field evaluation board, every Flight Surgeon should carefully review Paragraph C-7318 of the BUPERS Manual. A copy may be obtained from the Administrative Officer of any Navy command.

Special Board of Flight Surgeons

At times, questions arise as to a man's fitness to fly which become quite complex in terms of the issues involved. The commanding officer of the individual concerned, on the advice of the local Flight Surgeon, will request examination by the Special Board of Flight Surgeons. In this event, the referring command will submit to the Chief, Bureau of Medicine and Surgery, completed Standard Forms 88 and 89 with appropriate clinical addenda and other relevant information. The case then is reviewed by the Advisory Council for Aerospace Medicine. In questionable cases which cannot be resolved with the information supplied, a recommendation is made to the Chief of Naval Personnel or to the Commandant of the Marine Corps that the individual be ordered to report to the Naval Aerospace Medical Center to appear before the Special Board.

Additional means whereby individuals are referred to the Special Board are outlined in BUMED Instructions of the 6410.2 series. These deal generally with the problem of flight trainees and other aviation personnel in the Pensacola area.

The Special Board of Flight Surgeons consists of the Commanding Officer and senior members of the Naval Aerospace Medical Institute, all resident Flight Surgeons attached to the Institute, and appropriate specialists from the Naval Hospital of the Naval Aerospace Medical Center. The Board convenes each Friday to review cases for which all relevant information is available. The individual will appear before the Board at this time.

The individual concerned has a right to appeal the decision of the Board. In such an event, a Board of Senior Flight Surgeons may be convened to review the case.

Board of Senior Flight Surgeons

For those cases in which appeal of a recommendation or decision concerning an individual's fitness to fly is requested by the Chief of Naval Personnel, or the Commandant of the Marine Corps in the case of Marine Corps personnel, a Board of Senior Flight Surgeons is convened at the Bureau of Medicine and Surgery. This board consists of five members, three of whom must be Flight Surgeons. Individuals appealing a decision, for medical reasons only, may request appearance before this board. Decisions of this board are final and may not be appealed further.

SPECIAL ASSIGNMENTS

There are a number of unique and challenging special assignments open to Flight Surgeons in naval aerospace medicine. These assignments offer an opportunity to participate in unusual medical research programs, to study human functioning under a variety of environmental conditions, and to deal with the most advanced medical research equipment. The national space program provides an excellent example. There are several billets in the National Aeronautics and Space Administration in which Navy Flight Surgeons can continue to contribute to the advancement of manned space flight. In reviewing prior Navy accomplishments in this field, it may be noted that the first spacesuit, used in Project Mercury, was a direct modification of the Navy Mark IV full pressure suit.

Unique research facilities are available within the Navy. At the Naval Medical Research Institute, basic research is conducted in problems related to aerospace medicine. At the Aerospace Crew Equipment Department, a Flight Surgeon may work with the world's highest ejection seat tower. At the Aerospace Medical Research Department, the world's largest human centrifuge is available in support of research programs.

Locales for possible assignments for Flight Surgeons cover the entire world. Navy Flight Surgeons now are on duty in the Antarctic, where they have played a major role in research in arctic medicine. Other oversea assignments include billets in Spain, Great Britain, Italy, Morocco, Iceland, Newfoundland, Japan, and other parts of the world. Assignments are available at air stations in the several States, including Alaska and Hawaii. A career Flight Surgeon may expect a succession of unusual and interesting assignments during which his travels may encompass most of the earth.

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CHAPTER 4

Programs and Facilities in Support of Aerospace Medicine

AVIATION PHYSIOLOGY PROGRAM
  Personnel
  Equipment and Training
  Squadron Support
AVIATION PSYCHOLOGY PROGRAM
NAVAL AIR SYSTEMS COMMAND
NAVAL AVIATION SAFETY CENTER
  Human Error Research and Analysis Program (HERAP)
AEROSPACE CREW EQUIPMENT DEPARTMENT
AEROSPACE MEDICAL RESEARCH DEPARTMENT
ARMED FORCES INSTITUTE OF PATHOLOGY

WITHIN THE Navy, there are a number of programs and facilities which support the practice of aerospace medicine. These offer services which can help the Flight Surgeon to achieve and maintain operational readiness among his aviators. He should, therefore, understand the mission and particular capabilities of each such organization.

Communications.—The Flight Surgeon deals directly with the Armed Forces Institute of Pathology in cases requiring emergency examination of tissue or information regarding aviation pathology issues. He deals directly with Aviation Physiology Training Units regarding training schedules. In dealing with other organizations, however, such direct communication is neither appropriate nor efficient.

For medical and administrative matters, the Flight Surgeon should submit his request for information or services through official channels to:

Chief, Bureau of Medicine and Surgery (Code 5)
Navy Department
Washington, D.C. 20390

Personnel at the Bureau, being conversant with the activities of other Navy organizations and field units, will refer inquiries to that facility most qualified to deal with the issue.

For matters dealing with aviation safety and survival equipment, inquiries should be made through official channels to:

Naval Air Systems Command Headquarters (Code AIR 531)
Navy Department
Washington, D.C. 20360
or to
Naval Aviation Safety Center
Naval Air Station
Norfolk, Va. 23511

Again, such inquiries will be referred to the appropriate facilities. On occasion, operational problems concerning correct usage of equipment, need for modification, and proper functioning of on-hand equipment, require immediate decisions. Based on operational urgency, a dispatch or telephone contact is appropriate if authorized by the commanding officer.

AVIATION PHYSIOLOGY PROGRAM

The Naval Flight Surgeon is charged with the responsibility of insuring that all flight personnel under his jurisdiction are well informed regarding the physiological stresses imposed by modern aviation and thoroughly familiar with the personal protective equipment available to them. To assist the Flight Surgeon in this mission, the Navy has established a program of physiological training.

The OPNAV Instruction of the 3740.3 series entitled "Aviation Physiology Training/Indoctrination" defines the duties of aviation personnel with respect to physiological training. The Aviation Physiology Training Unit is an important link in this program and in the operational readiness of the Fleet. This unit helps the Flight Surgeon keep his pilots trained to cope with the many stresses encountered in flight. There are fourteen such units in the U.S. Navy, operating at the following locations:

Naval Air Station, Quonset Point, R.I.
Naval Air Station, Norfolk, Va.
Marine Corps Air Station, Cherry Point, N.C.
Marine Corps Air Station, Beaufort, S.C.

Naval Air Station, Cecil Field, Fla.
Naval Aerospace Medical Institute, Pensacola, Fla.
Naval Air Station, Corpus Christi, Tex.
Marine Corps Air Station, El Toro, Santa Ana, Calif.
Naval Air Station, Lemoore, Calif.
Naval Missile Center, Point Mugu, Calif.
Naval Air Station, Miramar, Calif.
Naval Air Station, Whidbey Island, Oak Harbor, Wash.
Naval Air Station, Barbers Point, Oahu, Hawaii.
Naval Air Test Center, Patuxent River, Md.

Personnel

The billet structure of an Aviation Physiology Training Unit is shown below.

	Designator/NEC	*Number*
Aviation Physiologist	2300	2
Aviation Physiology Technicians	8409 (HM)	4–10
Aviation Medicine Technicians	8406 (HM)	1–2
Parachute Riggers	7312 (PR)	1–2
Tradevmen	7533 (TD)	1–2

The Aviation Physiologist, working with Flight Surgeons, conducts a program which includes prophylaxis of certain aviation problems frequently encountered and potentially debilitating; familiarization with the various types of oxygen breathing equipment; the requirements for and the hazards of oxygen; cabin pressurization and the potential problems due to loss of pressurization; personal airborne protective equipment; problems of night vision and/or flash blindness; and the physiological aspects of emergency egress systems.

Aviation Physiologists are officers of the Medical Service Corps whose academic background and naval training have prepared them for aviation physiological training assignments. These officers, many of whom have earned advanced degrees, are given special training and flight indoctrination at the Naval Aerospace Medical Institute in Pensacola.

Aviation Physiology Technicians are selected Hospitalmen who have undergone 16 weeks of training (also at the Naval Aerospace Medical Institute) in aviation physiology and the operation of support training aids. These technicians operate low pressure chambers, ejection seat trainers, other training aids, fit and adjust aviators' protective equipment, and deliver lectures in certain areas of aviation physiology. Their sea duty assignments are usually to squadrons on aircraft carriers.

Equipment and Training

An important item of equipment at an Aviation Physiology Training Unit is the low pressure chamber. There are three basic types of chamber flights: (a) altitude indoctrination; (b) pressure breathing; and (c) full pressure suits.

An altitude indoctrination flight includes a preflight briefing, the fitting of oxygen masks, seating of personnel inside the chamber, a preflight check of the oxygen equipment, and a simulated ascent to 30,000 feet. At 30,000 feet, trainees remove their oxygen masks to experience the effects of hypoxia with its progressive loss of psychomotor efficiency. Masks then are replaced and a descent made to ground level pressure. Pressure breathing flights involve ascent to a simulated altitude of 43,000 feet for a demonstration of the effects of pressure breathing on the respiratory system and the actual mechanics of pressure breathing. Full pressure suit flights involve ascents to 70,000 feet for a demonstration of the functioning of the suit, the range of mobility that may be realized while wearing this garment, and an explosive decompression from 30,000 feet to between 50,000 and 60,000 feet in 500 milliseconds to illustrate the protective features of the suit in the event cabin pressurization is lost. Instructions regarding the various types of flights may be found in the Utilization Handbook for the specific low pressure chamber installed at the Aviation Physiology Training Unit.

Figure 4-1 shows the low-pressure chamber (Device 9A9) located at the Naval Air Station, Cecil Field. Ample viewing areas are provided to enable the Flight Surgeon and other personnel to monitor activities within the chamber during training sessions. Figure 4-2 shows personnel within a low pressure chamber prepared to begin a training session.

The Aviation Physiologist insures that proper instruction is given in the use of oxygen equipment and that trainees demonstrate their knowledge of the equipment during the actual low pressure chamber flight. Pilots are encouraged to use their own personal equipment, thus enabling the Aviation Physiologist and the Flight Surgeon to observe and correct dangerous habits that the trainee may have developed during flight operations.

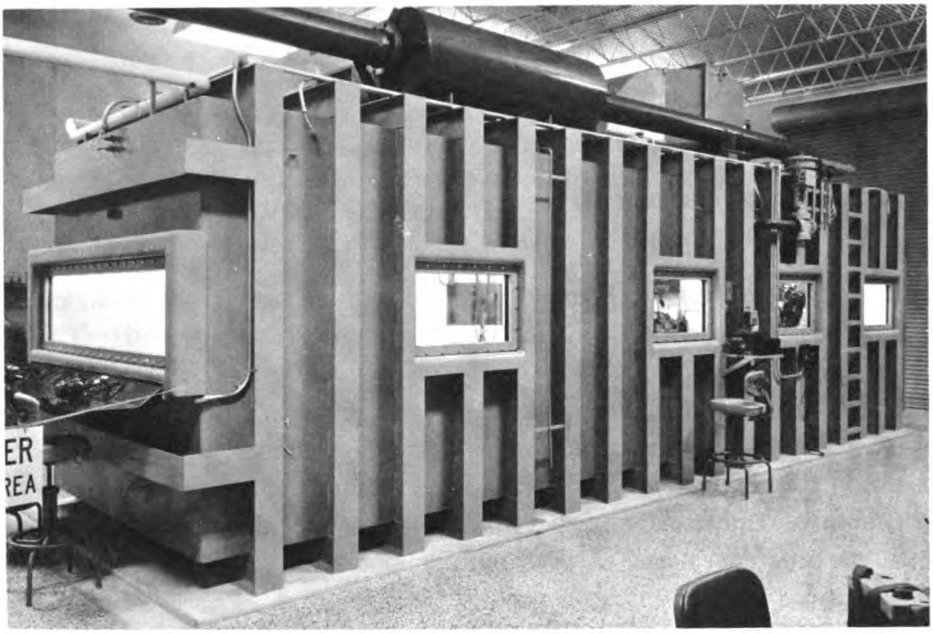

FIGURE 4–1. *Low pressure chamber (Device 9A9) at NAS, Cecil Field, Florida.*

A typical Aviation Physiology Training Unit has the following equipment:

- Low Pressure Chamber
- Ejection Seat Trainer
- Night Vision Trainer
- Static Ejection Seat Displays
- Oxygen Regulators (all types)
- Aeromedical Training Films
- Full Pressure Suit Training Facilities (selected units)
- Flash Blindness Indoctrination Trainer (selected units)

In addition, some units furnish water survival training and have at the training pool the following equipment:

- Dilbert Dunker
- Parachute Drag Trainer
- Tower Jump
- Helicopter Rescue Devices

A typical schedule for refresher training of aviators consists of lectures concerning aviation physiology, oxygen equipment of the type in the aircraft being flown by the pilots, and appropriate ejection seat training. A low pressure chamber flight and practice ejection on the ejection seat trainer are

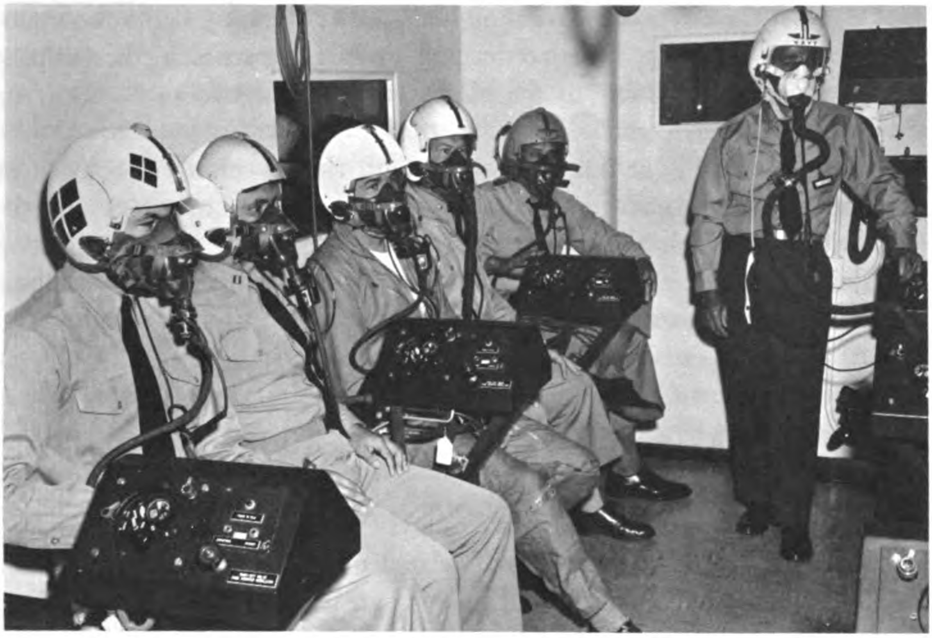

FIGURE 4–2. *Training session in low pressure chamber.*

included. Night vision refresher training or flash blindness indoctrination might be offered the same day. Water survival training, if available, would probably be scheduled on a separate day due to the length of training.

Squadron Support

A number of additional topics falling within the purview of aviation physiology are discussed by the Flight Surgeon during his squadron lectures. These include sensory illusions of flight, physiological effects of high-intensity noise, hearing-conservation measures, antiexposure suits, acceleration effects, anti-G suits, protective helmets, restraining devices, parachutes and harnesses, flotation gear, ditching procedures, survival equipment, emergency egress systems, new protective and safety equipment, and additional full pressure suit training.

Personnel of the Aviation Physiology Training Units periodically visit individual squadrons for discussions with safety officers, survival officers, personal equipment officers, and aircraft maintenance officers. These discussions represent an excellent means of coordinating squadron training with that given at the Physiology Training Units. The Training Units also maintain a stock of current films concerning various aerospace medical topics. These are easily accessible to the Flight Surgeon assigned to a Naval Air Station having such a unit.

Normally, the Flight Surgeon works with the Aviation Physiologist in presenting certain topics to squadron personnel at safety meetings and special briefings. Close liaison between the Flight Surgeon and the Aviation Physiologist greatly enhances the squadron's overall knowledge, operational readiness, and performance.

AVIATION PSYCHOLOGY PROGRAM

The Aviation Psychology Program employs experimental psychologists who work on basic and applied research related to human performance in the military environment. These psychologists are assigned to major aerospace medical research facilities in the Navy, where they work closely with medical personnel.

A major area of the Aviation Psychology Program concerns the selection of naval aviators. At one time, the Chief of Naval Air Training estimated that the use of Navy and Marine Corps Aviation Selection Tests saved about $9 million annually in training costs.

During peacetime, the Navy conducts one of the largest educational and training programs in the country. Much of the work of Aviation Psychologists is directed toward improving the efficiency of this massive training effort. Their research is concerned not only with theoretical implications of the learning process, but also with problems of syllabus analysis, retention of little-used skills such as those required to deal with inflight emergencies, fatigue effects, and the influence of various psychological and physiological stresses on the efficiency of learning. Research is also concerned with evaluation of new techniques such as closed-circuit television and computer-assisted instruction.

Aviation psychology also contributes to effective military programs through appropriate engineering design—or human engineering—of the man-machine interface of weapon systems. Such efforts attempt to define the optimum utilization of the human as an integral link within a military system. Workspace must be designed to sustain reliable performance under conditions of stress and fatigue and display inputs must present information in a manner which will optimize the control and decision-making functions of the operator.

A meaningful human engineering program requires precise information concerning human performance capabilities. Since a considerable amount of information remains to be defined with precision, Aviation Psychologists are frequently engaged in very basic research within the field of psychophysiology. Such research attempts to chart the limits of human sensory and perceptual capacities.

Aviation Psychologists also conduct small-group research, concerned principally with the problem of maximizing group productivity. The effectiveness of small military units generally is based upon their internal organization. The search, then, is for mechanisms which enhance or detract from group performance, based upon consideration of personality variables, emergent leadership phenomena, group cohesiveness under stress, and similar social variables.

Indoctrination and training of Aviation Psychologists is accomplished at the Naval Aerospace Medical Center, Pensacola. The flight portion of their training is identical to that given Flight Surgeons, and they receive a similar flight designation at its conclusion. They may expect to serve at one of many Navy aerospace research facilities as contributors to the Navy's high standards of performance in aerospace operations.

NAVAL AIR SYSTEMS COMMAND

The Naval Air Systems Command is responsible for the development of Navy aircraft and associated components, including safety and survival equipment for aviators. Figure 4–3 shows the organization of this command. Within this Command, of special interest for a Flight Surgeon, is the Crew Systems Division which is responsible for the development and continuing improvement of such items of personal protective equipment as flight clothing, full pressure suits, protective helmets, oxygen systems, and flotation devices.

In the development of personal protective equipment, it is the intent of the Naval Air Systems Command that whenever possible the equipment will increase the military effectiveness of the weapon systems with which it operates and by so doing contribute both to combat readiness and to an increased level of safety. The purpose behind personal protective equipment thus is

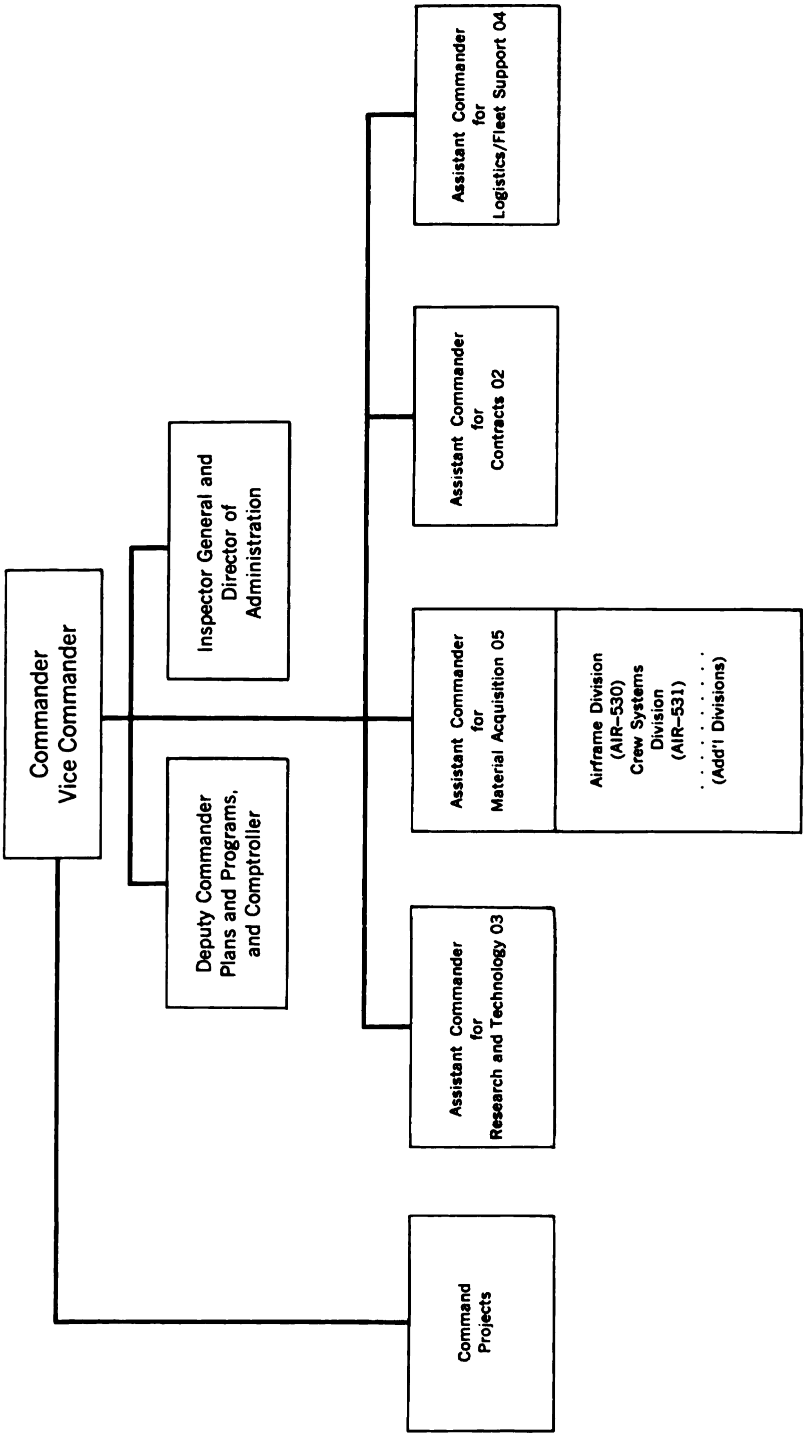
Commander
Vice Commander
Inspector General and Director of Administration
Deputy Commander Plans and Programs, and Comptroller
Command Projects
Assistant Commander for Research and Technology 03
Assistant Commander for Material Acquisition 05
Airframe Division (AIR-530)
Crew Systems Division (AIR-531)
. . . .
(Add'l Divisions)
Assistant Commander for Contracts 02
Assistant Commander for Logistics/Fleet Support 04

FIGURE 4-3. *Organization of the Naval Air Systems Command, showing units of principal interest to a Flight Surgeon.*

not necessarily to provide total protection, but rather to provide the maximum protection consistent with the mission of the man and aircraft. In so doing, the following three rules are observed:

1. The equipment should reduce the hazard associated with a particular occupation or mission.
2. The equipment should be constructed in such a manner as to develop a sense of confidence in the reliability of the device as far as aircrewmen are concerned.
3. Use of the equipment should not impair the effectiveness or efficiency of the weapon system.

To accomplish its mission, the Naval Air Systems Command has a number of field installations and laboratories under its jurisdiction.

As an example, when an operational command informs NAVAIRSYSCOM of a requirement for equipment development, the problem is passed on to the appropriate field laboratory. If the requirement is for a piece of personal equipment, it is passed to the Naval Air Engineering Center in Philadelphia. The Center then passes it on to the Naval Aerospace Crew Equipment Department, where the Director is a Naval Flight Surgeon, even though the laboratory is a NAVAIRSYSCOM facility. ACED performs the required research, development, test, and evaluation and reports to NAVAIRSYSCOM. This in turn is followed by a field test and/or an evaluation of the laboratory recommendation, performed by the Naval Air Test Center at Patuxent River, Md. When favorably reported on by the latter activity, NAVAIRSYSCOM arranges the preparation of a Military Specification, which is used in obtaining competitive bids, and the letting of a procurement contract on the basis of these bids. Thereafter, the equipment is distributed by the supply organization.

NAVAL AVIATION SAFETY CENTER

In 1951, the Flight Safety Branch of the Office of the Chief of Naval Operations was moved from Washington, D.C., to Norfolk, Va. The same year, it was redesignated the Naval Aviation Safety Activity. Its total strength at that time was 27 officers, enlisted, and civilian personnel, and its major activities were recordkeeping and the preparation of accident statistics. In early 1955, the Activity was redesignated the Naval Aviation Safety Center, which in 1957 became a flag command. During this period, the assigned mission was expanded to "assist the Chief of Naval Operations in all phases of aviation safety for the formulation of policies necessary to maintain the highest practicable level of combat readiness."

As the field representative and staff arm of the Chief of Naval Operations for all matters pertaining to aviation safety, the Safety Center serves as a clearinghouse for safety material and a reference center for all Aviation Safety Officers. The Center provides a continuous opportunity for ASOs to obtain safety information and exchange ideas with their colleagues directly and via *Approach* and other Center publications.

In addition to daily efforts in the interest of accident prevention, some recent programs initiated by the Center include:

1. Increased visits and liaison trips to operating units to provide a better understanding of the magnitude of the safety problem, and to exchange and discuss vital safety information relative to type aircraft in the operational environment.

2. Conferences with manufacturers of each major aircraft in the inventory to bring together the full experience of the Fleet, the Center, and the design safety team of the manufacturer to solve existing problems.

3. Augmenting the Aviation Safety Officer training and education program of the University of Southern California by providing a special five-day safety course at the Center. This provides a broad and practical program for new Aviation Safety Officers and serves as a refresher to more experienced officers.

Figure 4-4 shows the organization of the Naval Aviation Safety Center. Of particular interest for Flight Surgeons is the Aeromedical Department, directed by a Flight Surgeon with the rank of captain. The staff of this department includes other Flight Surgeons and an Aviation Pathologist. The chief concern of the Aeromedical Department is with the people of naval aviation: the pilots who fly the planes and the men in hundreds of different jobs who keep them flying. A primary source of information for this department is the Medical Officer's Report of Aircraft Accident, completed by the investigating Flight Surgeon and made a part of every Navy aircraft accident report.

The Biophysics and Survival Division of the Aeromedical Department deals with personal survival equipment, escape systems, and land and water survival procedures. Division personnel monitor the daily message traffic from the Fleet for actual and potential problems in these areas. Efforts

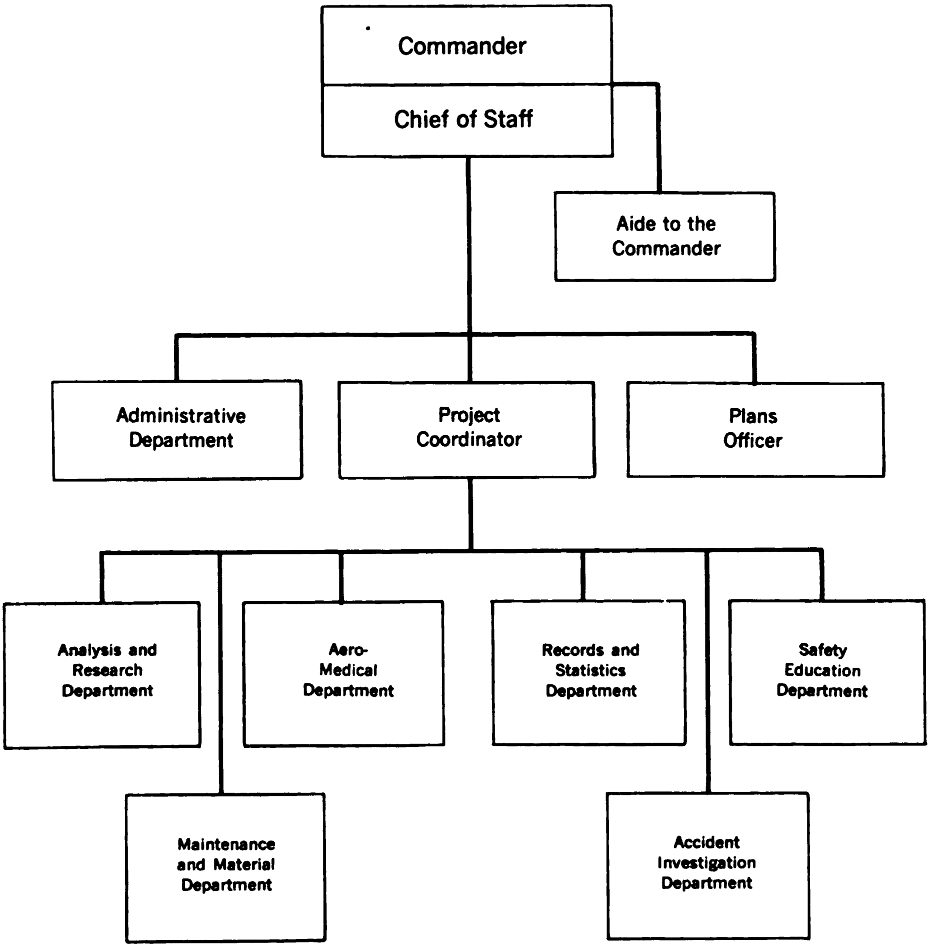
Commander
Chief of Staff
Aide to the Commander
Administrative Department
Project Coordinator
Plans Officer
Analysis and Research Department
Aero-Medical Department
Records and Statistics Department
Safety Education Department
Maintenance and Material Department
Accident Investigation Department

FIGURE 4-4. *Organization of Naval Aviation Safety Center.*

of this division have resulted in substantial improvements in pilot and aircrew protective equipment.

The Biomedical Division studies physiological and pathological conditions of pilots and crewmen before and after accidents. The Environmental Health Division and the Aviation Pathology Division complete the structure of the Aeromedical Department.

Safety information developed by the Aeromedical Department is disseminated through the following channels:

1. Flight Surgeon's Newsletter.
2. Personal/Survival Equipment and Escape Crossfeeds.
3. Escape and Rescue Summaries.
4. Special studies and articles for *Approach*.

Human Error Research and Analysis Program (HERAP)

The efforts of the Safety Center have stimulated a number of new Navy programs in recent years, all designed to effect an improvement in the safety record. Some of these are Navywide recognition of the impact of accidents, establishment of the replacement pilot training program, initiation of procedures for detection of incipient engine failures and other engine reliability programs, retrofit of low level ejection capability seats in all firstline aircraft, procedures designed to reduce wheels-up landings, and utilization of field arresting gear. As a result of these programs, the all-Navy aircraft accident rate has dropped from 5.4 in 1952 to 1.26 per 10,000 flight hours in 1966. Figure 4-5 shows the regular progression of this improvement in the safety record.

Inasmuch as human-error cause factors were assigned to 72 percent of all naval aircraft accidents during the period 1955 to 1964, it is obvious that any further reduction in the accident rate must focus on these human variables. To study accident causes and results in the human factors field, the Human Error Research and Analysis Program (HERAP) was established. This is a longitudinal systems analysis of human factors now assigned as causative agents. The study places emphasis upon the human element but at the same time integrates this emphasis with current approaches to equipment failure. The purpose of the program is to obtain and use appropriate information concerning human engineering as related to cockpit design and layout, as well as lighting, communications, training,

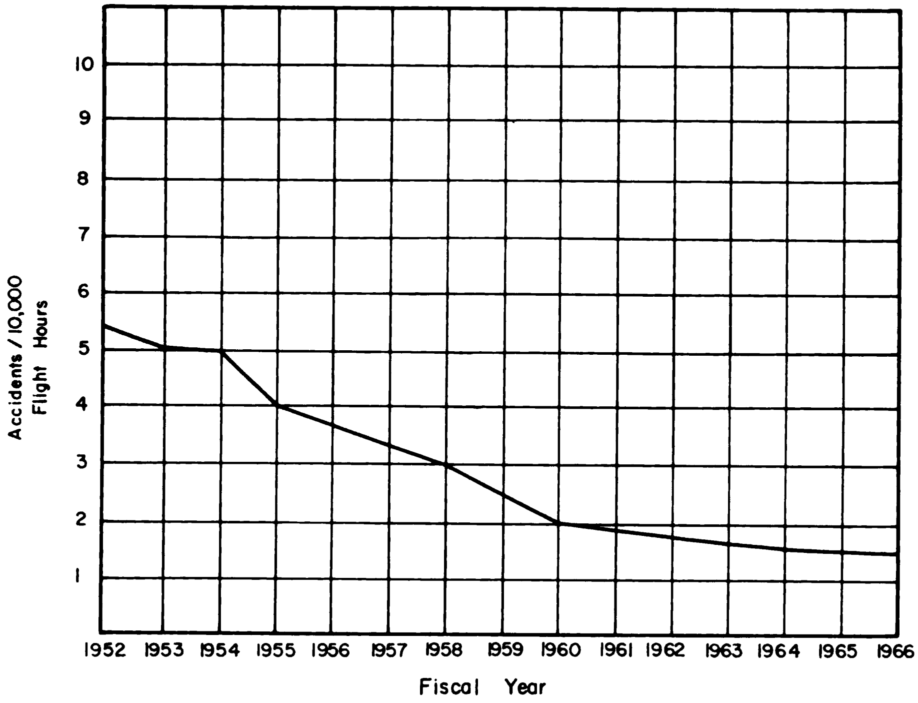

FIGURE 4-5. *All-Navy aircraft accident rate, FY's 52-66.*

personnel selection and assignment, and the total flight/operation environment and the interface between man and machine.

AEROSPACE CREW EQUIPMENT DEPARTMENT

The Aerospace Crew Equipment Department, formerly the Aerospace Crew Equipment Laboratory, is a part of the Naval Air Development Center, Johnsville, Warminster, Pa.; however, it is physically located at the Naval Base, Philadelphia, Pa. The Laboratory originated in 1942 as the Controlled Elements Group of the Aeronautical Materials Laboratory. The Group consisted of four engineers and was primarily concerned with the evaluation of oxygen equipment and flight clothing. Since then it has grown into an organization employing over 100 naval officers, enlisted personnel, civilian scientists, technicians, and support personnel. The mission of ACED is to conduct research, development, test, and evaluation of aerospace crew equipment, ranging from pen-type flare guns to full pressure suits capable of sustaining life in the space environment.

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The facilities of ACED include many items of research and testing equipment which are not available at any other military facility or in any industrial plant. Use of these facilities allows realistic stresses to be applied to both personnel and equipment and thus insures that items of protective equipment will be appropriate for the operational environment in which they will be used. Test facilities located at ACED include:

1. Three personnel-type altitude chambers providing a combined capability for stimulating altitudes of 100,000 feet, temperatures of +280° F to −70° F, explosive decompression times of 0.05 second, and precise control of relative humidity. The altitude/thermal/decompression chamber, which is part of the complex, is capable of space and orbital environmental simulation with respect to altitude, temperature, and air quality and quantity.

2. A Bioastronautics Test Facility which permits confinement in isolation of one to six volunteers under simulated space capsule conditions for indefinite time periods at any simulated altitude from sea level to 100,000 feet. Unique systems for oxygen production, reclamation, and air purification are incorporated. Complete feeding, sanitary, and sleeping facilities, and simulated work consoles are provided. A model of this facility is shown in figure 4–6.

3. An Ejection Seat Test Tower which is the tallest in the world. This tower is 150 feet high and is capable of ejecting weights of up to 600 pounds for human and equipment investigations.

4. A Vertical Accelerator of the drop tower variety. This device is 141 feet high and is capable of giving accelerations commensurate with peak dynamic loadings of 80,000 pounds. Figure 4–7 shows the Vertical Accelerator and the Ejection Seat Test Tower.

5. An Underwater Test Facility, 32 feet in diameter and 30 feet deep. The capacity of this facility allows the simulation of escape from sinking aircraft or spacecraft. Detailed studies can be conducted of emergency underwater escape systems.

6. A Flexible Cockpit Test Device capable of simulating or developing any aircrew workspace by allowing spatial relocation of components within a work console complex.

7. A Linear Accelerator for crash and crew restraint programs capable of accelerating 2,000 pounds with a force of up to 43 G. The maximum sled velocity is 140 feet per second. Figure 4–8 shows the test firing of a developmental rocket escape system from the jet-propelled sled.

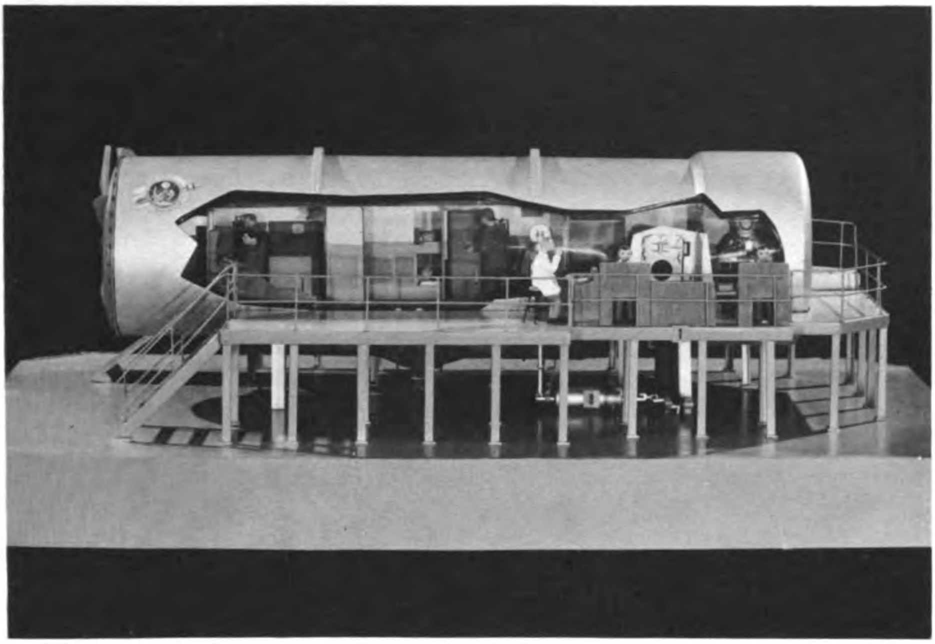


FIGURE 4-6. *Bioastronautics Test Facility at ACED.*

The Aerospace Crew Equipment Department is credited with many notable accomplishments relating to the protection of aircrewmen. Aircraft emergency escape systems, with ground level capability, have been developed. Criteria for underwater escape from aircraft have been studied and appropriate design specifications for escape equipment established. Trajectory patterns, design criteria, and evaluation methods for the ejection seat escape systems developed in ACED have been adopted by industry.

Advanced-design, constant-wear, and quick-don-type exposure suit systems have been developed and placed into Fleet use. In cooperation with other Government agencies, work to determine the effect of high thermal pulses on various materials for special weapons protective clothing has been coordinated. Special aircrew protective systems have been developed using these materials.

Human engineering concepts for aircraft controls and interior and exterior lighting of aircraft have been developed. Studies have been conducted of the legibility of instrument data presentations under normal and emergency conditions. These studies have defined optimum markings, shapes, and locations of flight controls.

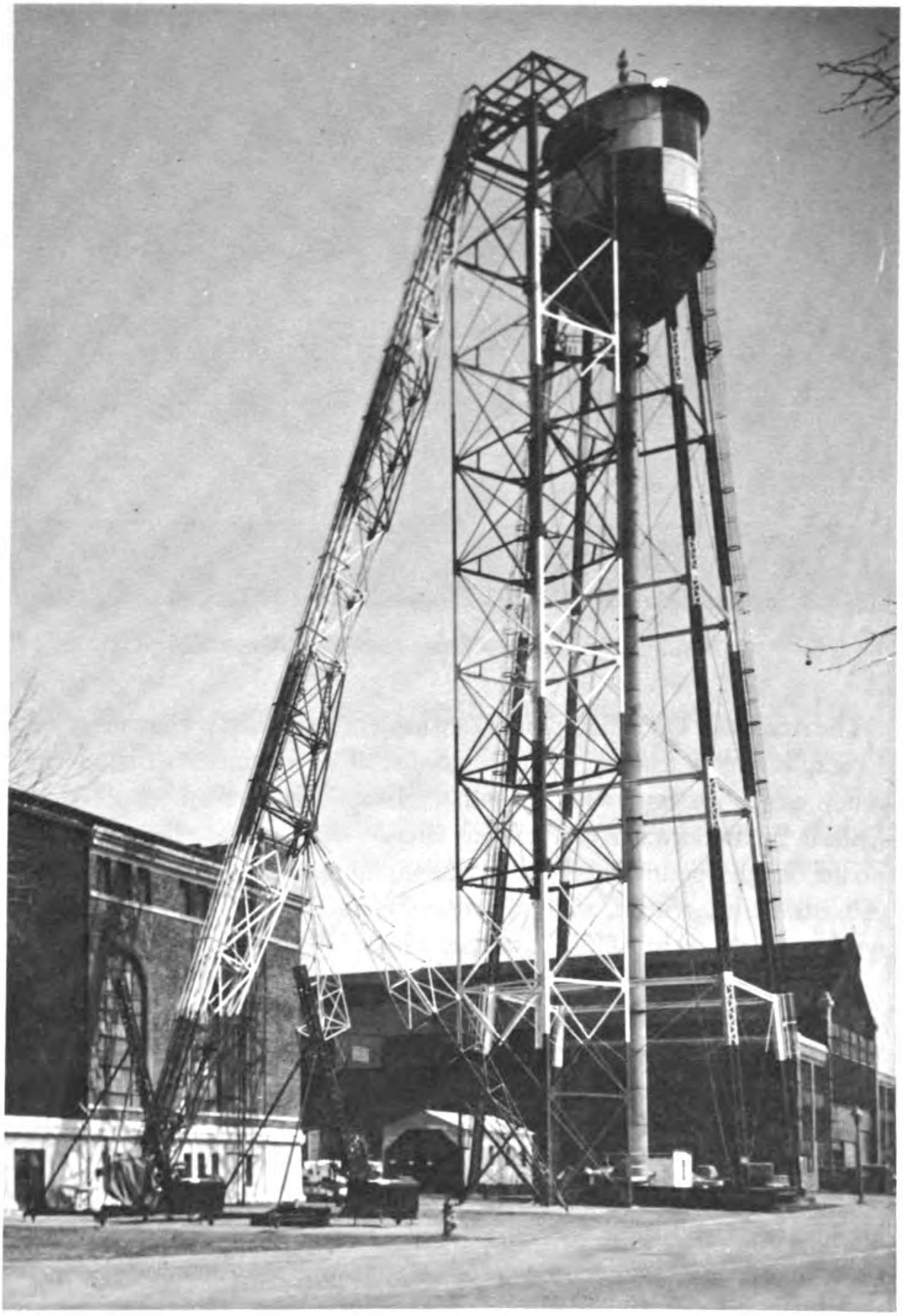


FIGURE 4–7. *View of ejection seat test tower (on left) and vertical accelerator at ACED.*

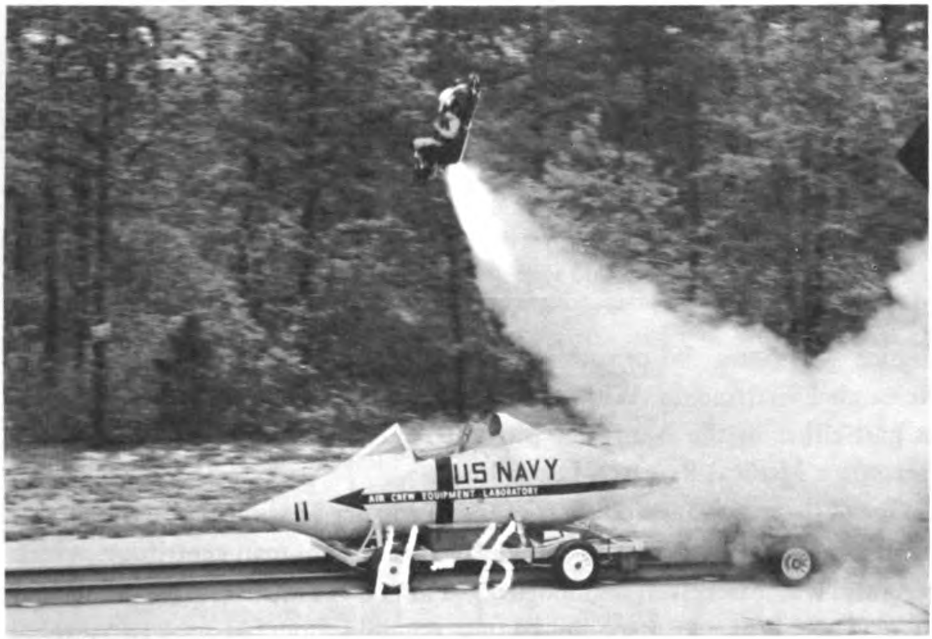


FIGURE 4–8. *Test firing of a developmental rocket escape system from jet-propelled sled.*

The Navy entered the NASA Mercury Project in July 1959, when a modified version of the Mark IV full pressure suit, developed at ACED, was selected for use by Mercury astronauts. This suit was designed to operate at a 5 psi absolute pressure, using a single-gas system with oxygen for both ventilation/pressurization and breathing, thereby providing a simplification of earlier pressure suits developed at ACED.

AEROSPACE MEDICAL RESEARCH DEPARTMENT

The Aerospace Medical Research Department (formerly the Aviation Medical Acceleration Laboratory), a part of the Naval Air Development Center, Johnsville, Warminster, Pa., is concerned primarily with the medical and physiological problems arising from the stresses of modern aviation and space activities. The charter of the Aerospace Medical Research Department states that it shall:

> Conduct research in the general field of aviation and space medicine, physiology, biophysical effects of acceleration forces, and human factors in

> aerospace engineering; develop, improve, test, and evaluate aviation and astronautic personnel equipment and related aircraft and spacecraft components. Provide residency training in aviation and space medicine for assigned personnel. Foster close working relationships with members of NAVAIRSYSCOM and BUMED in order to assure technical effectiveness. Provide consulting and advisory services to NAVAIRSYSCOM and BUMED, other Government activities, and Government contractors. Conduct contract placement, management, and technical supervision of R&D development projects and programs for components and systems. Prepare specifications for systems and equipment.

Figure 4–9 shows the organizational structure of the Aerospace Medical Research Department. Within this structure, the various divisions operate as part either of the Aerospace Medical Acceleration Laboratory or of the Aerospace Medical Research Laboratory.

One of the major areas of effort of the Aerospace Medical Research Department is stress physiology. In support of this is its human centrifuge, which recently was extensively remodeled. The new centrifuge has a 50-foot tubular-steel arm and a spheroidal-type gondola mounted in a two-gimbal support. At a 50-foot radius of operation, a maximum of 40 G can be obtained at a rate of 10 G per second to 15 G, and 5 G per second thereafter. At a 22-foot radius of operation, a maximum of 100 G can be obtained. The gondola payload in the centrifuge is 1,000 pounds or 40,000 G-pounds. Figure 4–10 shows the centrifuge with the gondola located at the end of the supporting steel arm. Figure 4–11 shows an astronaut entering the gondola for a test of his capabilities to perform under the acceleration stresses of space flight.

Some of the more significant features of the modernized centrifuge are an interchangeable capsule concept which will minimize downtime between programs; a new centrifuge arm and gondola capable of accommodating larger and heavier payloads; new slipring stacks at all axes capable of transmitting more physiological, psychological, and engineering data to and from the gondola during programs; an increased number of rotary joints capable of commuting conditioned air, vacuum hydraulic fluids, and compressed air to the gondola; and increased programming and control capabilities of the centrifuge and gimbal drive motors. Of particular importance is the fact that the centrifuge can be operated as a "closed-loop" device in which the G forces applied to the centrifuge gondola are a function of the specific control actions of the subject inside the gondola.

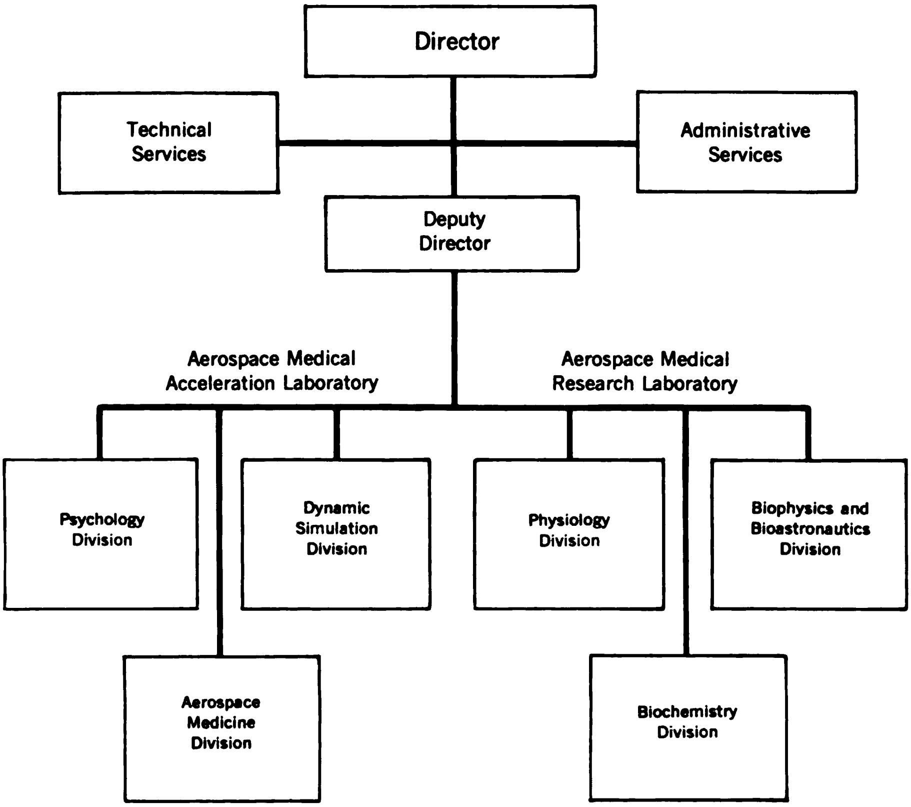

Director
Technical Services
Administrative Services
Deputy Director
Aerospace Medical Acceleration Laboratory
Aerospace Medical Research Laboratory
Psychology Division
Dynamic Simulation Division
Physiology Division
Biophysics and Bioastronautics Division
Aerospace Medicine Division
Biochemistry Division

FIGURE 4-9. *Organization of Aerospace Medical Research Department.*

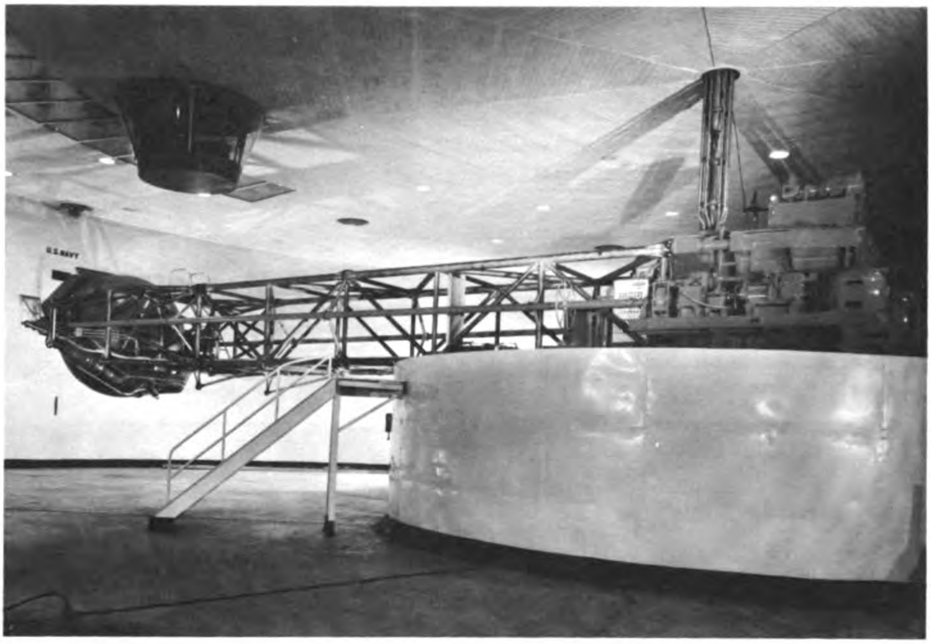


FIGURE 4–10. *AMRD human centrifuge.*

As used by the Aerospace Medical Research Department, the centrifuge provides a means of studying problems basic to all of medicine and pathology. For example, this device allows an assessment of the interaction between the cardiovascular system and the brain at various levels of acceleration stress. Inasmuch as the unconsciousness resulting from high acceleration forces is a function of a decreased cerebral oxygen supply, the centrifuge tests lead into other studies of ways in which the organism might use its oxygen supply more efficiently, or in which it might delay use of oxygen for organs of lesser importance than the brain. Acceleration stress thus is not considered unique, but rather as forming a part of the total field of biological stresses.

Many studies at AMRD are concerned with stresses other than those imposed by acceleration. For instance, the Thermal Branch of the Biophysics and Astronautics Division conducts research and development of nonflammable and fire-retardant materials for aviators' clothing as well as basic research in the field of the physiology of thermal burns. The Nomex fiber, now used in summer flight clothing for aviators, was perfected by personnel within this branch.

The Psychology Department conducts studies of human behavior, particularly as it relates to the stresses of the aerospace environment. Many of

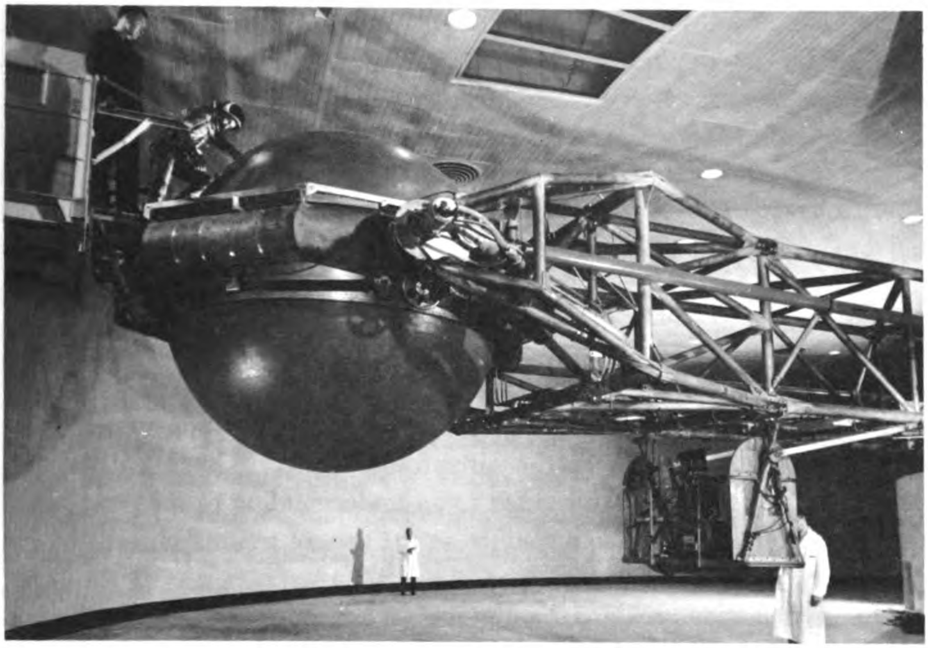


FIGURE 4–11. *Astronaut preparing for test in AMRD human centrifuge.*

these studies are concerned with the man-machine interface problems in new military systems. In the course of accomplishing these studies, it has been found that use of the human centrifuge as a true dynamic flight simulator often will reveal significant design problems which are not apparent through static simulation procedures. Basic studies of the Psychology Department are concerned with human information-processing capabilities and the essential elements of visual information.

Much recent work of the Aerospace Medical Research Department has been in support of development efforts in the space program. Launch and reentry profiles for both Mercury and Gemini missions have been simulated in the centrifuge. The ability of astronauts to control the spacecraft while wearing full pressure spacesuits was assessed through use of these acceleration profiles.

Under an agreement reached in 1950, the Aerospace Medical Research Department has a rather unique affiliation with the University of Pennsylvania. The agreement provides that the Director of Research of AMRD is nominated by the faculty of the School of Medicine of the university and holds a professorship in an appropriate department of the school. Other members of the professional staff of AMRD have been nominated and hold similar appropriate appointments in the school. As a result, the special facilities of AMRD, not duplicated elsewhere in the world, are available to the university for research purposes. Meanwhile, the full educational and consultative facilities of the university are open to the AMRD staff.

ARMED FORCES INSTITUTE OF PATHOLOGY

The introduction of commercial jets in British air transportation in 1954 was accompanied, unfortunately, by tragic results. The *Comet* accidents were investigated with extreme thoroughness, with much attention given to the condition of the remains of the aircraft occupants. The results of this pathologic investigation contributed to an understanding of the true nature of the structural defect which resulted in the type of trauma suffered by the passengers.

This investigation provided the stimulus for a meeting at the Armed Forces Institute of Pathology in March 1955, which was attended by aviation representatives of the Armed Forces of the United States, Canada, and the United Kingdom. The purpose of this meeting was to explore ways and means of counteracting the rising toll of aircraft accident fatalities in the three represented nations. As a result of the meeting, the Department of Defense directed the establishment of the Joint Committee on Aviation Pathology, with the AFIP as the central coordinating facility for the investigation of the pathology of fatal cases in aircraft accidents. The AFIP then established the Aviation Pathology Section, integrating it with the already existing Forensic Pathology Section. Because of the large number of cases, it became necessary in 1959 to reorganize the section, and a new division was created: the Military Environmental Pathology Division, with three branches, the Aerospace Pathology Branch, the Forensic Pathology Branch, and the Wound Ballistic Branch.

Because of the importance of biochemical support for the study of aircraft accident fatalities, a toxicology laboratory was established in the Aerospace Pathology Branch in 1956. This laboratory utilizes very sensitive tests for the analysis of blood and tissue to determine levels of toxic substances to which flying personnel might have been exposed.

AFIP personnel studying aircraft accidents have contributed a considerable amount of the literature relating pathology to aircraft accident fatalities.

Services of these personnel, and of the facility, are available to Flight Surgeons in support of their accident investigative efforts.

One of the great advantages of the Joint Committee and its operation is the fact that if any (participating) military service has a fatal accident near a medical facility of any other (participating) military service—regardless of the military service or country involved—the autopsy will be performed locally. As an example, U.S. Air Force pathologists in Europe provide pathologic support to the Royal Canadian Air Force, which has no pathologists stationed in Europe. Should an RCAF fatality occur in Europe, USAF pathologists at the medical facility nearest the accident will perform the autopsy and forward their findings to the AFIP for study. The AFIP, in turn, will send its report directly to responsible medical and aviation safety personnel of the country involved—in this case, Canada. This has proved a most effective method of submitting pertinent pathologic information promptly, without the delays usually associated with routine or "through channels" processing from one nation to another.

Recently a coding system has been completed by which medical data on aircraft accidents can be coded in a standardized fashion in the United States. This code has also been made available to the Canadian and United Kingdom aviation pathologists. Thus, for the first time, there will be established a standard repository and a system for coding medical data of this type. This code attempts to relate the flight profile and the aircraft to the individual fatality. It will be applied to case research in the future. The code is not cumbersome and was not developed for statistical analysis purposes but rather to assist physicians in using casefinding methods for presenting patterns of injury in individuals killed in aircraft accidents. It will also provide a method by which a long-term analysis can be carried out with respect to the importance of environmental factors and toxic substances in aviation safety.

CHAPTER 5

Aerospace Dentistry

THE FLIGHT SURGEON'S ROLE IN AEROSPACE DENTISTRY
AERODONTALGIA
ORAL HEALTH
PREVENTIVE DENTISTRY
DIAGNOSTIC PROCEDURES
    Diagnostic Procedures for Aerodontalgia
    Diagnostic Procedures for Medical-Dental Problems
DENTAL RECORDS AND TERMINOLOGY
    Designation of Teeth
    Restorations
    Missing and Replaced Teeth
    Abbreviations
REFERENCES

THE FLIGHT SURGEON'S ROLE IN AEROSPACE DENTISTRY

THE FIELD of aerospace dentistry or aerodontology requires a close working relationship between the Flight Surgeon and the Dental Officer. This relationship is necessary because aerospace dentistry is concerned with dental-medical ailments as well as disorders which are of a distinctly dental nature; and because dental treatments may have an adverse effect on the flight status of aviation aerospace personnel. All but the most unusual Navy and Marine installations to which the Flight Surgeon might be assigned will include personnel of the Dental Corps. Therefore, the Flight Surgeon need not concern himself with emergency dental procedures. These considerations are the basis for specifying the following dental-related duties for the Flight Surgeon.

1. *Medical Administrative Control.* The Flight Surgeon's responsibility for medical administrative control of flight personnel requires that he know which persons are undergoing dental treatment, the specific disorders being

treated, the particulars of the treatment being employed, and the effect of the treatment on the individual's fitness for flying duty.

2. *Diagnosis.* The Flight Surgeon may be required to interact with the Dental Officer in the differential diagnosis of medical-dental disorders. In unusual circumstances where a Dental Officer is not available, the Flight Surgeon may be called upon to diagnose and arrange for the disposition of emergency cases of dental and dental-related disorders.

3. *Dental Terminology and Records.* For two reasons, the Flight Surgeon should have a working familiarity with the terminology and records involved in aerospace dentistry. First, the interaction of the Flight Surgeon and the Dental Officer will be most effective when the Flight Surgeon has a knowledge of human dentition and associated terminology. Second, the Flight Surgeon is responsible for calling upon and coordinating the services of a Dental Officer for establishing or verifying the identity of deceased persons involved in fatal aviation accidents.

4. *Combat Duties.* As required during combat activity, Dental Officers discontinue definitive dentistry and assist the medical team in the care and treatment of the wounded. Generally, the Dental Officer directs his efforts toward: maxillofacial surgery, resuscitation and emergency anesthesia, and care of casualties. Aboard carriers, a Dental Officer is assigned to supervise one of the Auxiliary Battle Dressing Stations.

The content of this chapter has been selected to assist the Flight Surgeon in fulfilling these dental-related duties. Specifically, information is presented on aerodontalgia, dental hygiene, diagnostic procedures, and dental terminology and records.

AERODONTALGIA

Aerodontalgia is defined as toothache provoked by lowered barometric pressure during actual or simulated flight. Burket (1946) found that aerodontalgia is a condition intimately related to preexisting dental pathology and usually represents an acute exacerbation of subclinical symptoms. Orban and Ritchey (1945) state that changes in atmospheric pressure aggravate the impaired circulation in irritated or diseased pulps and may lead to early manifestations of inflammatory and degenerative changes in the pupal and periapical tissues. Armstrong and Huber (1937) state that the teeth are subjected to three distinct abnormal variations in environment during high-altitude flight: (1) decreased barometric pressures, (2) increased oxygen percentages, and (3) lowered atmospheric temperatures. Research has shown that these environmental factors have no adverse effect on normal teeth and dental restorations.

Early research (Sognnaes, 1947) also investigated radial acceleration as a possible activating factor in aerodontalgia. Flyers with a high incidence of dental and periodontal disorders were subjected to human centrifuge accelerations equal to and more than those experienced in flight. These experiments failed to reveal subjective symptoms referable to the teeth.

It has been suggested that air entrapped under restorations may be a cause of aerodontalgia. Numerous investigators have experimentally produced air bubbles under dental restorations and exposed the patients to low barometric pressure. No symptoms were experienced in these cases. This evidence, as well as rational analysis, indicates that trapped air under restorations is not a likely cause of aerodontalgia.

In summary, research indicates that the majority of cases of aerodontalgia are brought about by reduced barometric pressures causing circulatory changes in an already diseased or irritated pulp. Furthermore, treatment of aerodontalgia does not vary from therapy rendered for toothache at ground level.

ORAL HEALTH

The Flight Surgeon alone bears the ultimate responsibility for the overall health of the Air Wing to which he is attached. Often overlooked is his obligation in insuring adequate oral health as a part of the physical fitness of the aviator and other squadron personnel. It is essential that he have a firsthand knowledge of the oral health of those who look to him for guidance in obtaining proper medical treatment. Accordingly, it is essential that he have rapport with the Dental Officer at all times and seek dental treatment for those personnel who are his responsibility.

Dental treatment in the Navy is a deficit operation in that there are insufficient personnel and facilities to accomplish the mission in its entirety. All Navy and Marine Corps personnel do not at present receive an annual prophylaxis, or the completion of all operative, prosthodontic or periodontic treatment. In situations where the concept of "whole ship" or "whole squadron" dentistry is not feasible, the state may develop where only "the squeaking wheel gets the grease."

Many recruits and reservists coming to active duty have deplorable dental health. They average seven to eight carious lesions per man. During recruit training, approximately 25 percent of needed dental treatment is accomplished. Accordingly, many men reach their squadrons in dire need of further dental treatment. Furthermore, each squadron has its floating population which avoids dental treatment because of apprehension and lack of oral health education. For example, one survey demonstrated that 43 percent of the entering recruits at a training station did not own a toothbrush. Consequently, each squadron has a large number of potential dental casualties.

Numerous problems exist in providing squadron personnel with complete dental treatment. An unrealistic Dental Officer/population ratio is basic but not insurmountable. Seemingly, another major problem attendant to the lack of dental treatment for squadron personnel is insuring their presence at the dental clinic with sufficient leadtime to accomplish the necessary treatment prior to deployment. Advance planning on the part of Medical Department representatives would do much to overcome this hurdle.

All dental records are color coded in accordance with the dental requirements of the individual; viz, Class I: no treatment needed; Class III: urgent treatment needed. Accordingly, a review of the records well in advance of known deployments is a responsibility of the Medical Department representative. He should realize that a Class I can degenerate into a Class III without visible evidence in the files. Hence, the additional responsibility of insuring periodic dental examinations should be stressed.

The Flight Surgeon is urged to establish close liaison with the Dental Officer, both at his home station and while deployed. He should have a similar relationship with his Senior Petty Officer and the Senior Dental Technician. With such a working relationship, the creation of a protocol-generated barrier may be avoided. The Flight Surgeon can—and should—guarantee the participation of the personnel for whom he is responsible in the Preventive Dentistry Program conducted by the Naval Dental Corps.

PREVENTIVE DENTISTRY

Dental disease affects virtually 100 percent of military personnel. The two most common dental disorders, dental caries and periodontal disease, are usually the result of neglect and lead to needless discomfort, loss of productive time, and decreased unit effectiveness. Today there is sufficient knowledge of the nature and etiology of oral disease, and there are adequate techniques of prevention to rule out virtually all new dental caries and periodontal disease. Consequently, an organized program of preventive dentistry is conducted at all Navy and Marine Corps installations having dental personnel. The Flight Surgeon should familiarize himself with the contents of the Bureau of Medicine and Surgery *Preventive Dentistry Manual* (NAVMED 5087), the preventive dentistry program of the Navy or Marine Corps installation to which his unit is attached, and priority preventive dentistry measures that should be accomplished prior to deployment of his unit.

The Flight Surgeon can assist the Dental Officer by seeing that personnel have the following accomplished to the extent dental services permit:

1. An annual dento-oral examination to assess the status of the dentition, periodontium and other oral tissues, and oral hygiene.
2. An annual dental prophylaxis to include (a) removal of plaque, exogenous stains, and calculus from the teeth, and (b) application of an approved topical anticariogenic agent (three-agent stannous fluoride treatment).
3. Preventive dentistry counseling to individual patients and groups on:
    - *a.* Effective toothbrushing and oral soft tissue care at home.
    - *b.* The use of approved dental disclosing agents during home care procedures.
    - *c.* How to clean prostheses and maintain the supporting tissues in a healthy condition.
    - *d.* Diet, consistency of food, frequency of eating, and food habits for proper nutrition and dental health.
4. Construction of mouthguards for participants in contact sports. Recipients of mouthguards must be instructed concerning cleaning and maintenance procedures.

The Flight Surgeon should inspect the hard and soft tissue of the oral cavity as a matter of routine in all physical examinations, to insure that dental disease is detected in its incipient stage, during which period treatment is most effective.

DIAGNOSTIC PROCEDURES

The Flight Surgeon may be called upon to participate with the Dental Officer in the diagnosis of medical-dental ailments. Also, on rare occasions, it may be necessary for the Flight Surgeon to diagnose and arrange for the disposition of distinctly dental problems when a Dental Officer is not readily available. To assist the Flight Surgeon in preparing for these roles, the following summarizes diagnostic procedures, first for cases of aerodontalgia, and then for medical–dental disorders.

Diagnostic Procedures for Aerodontalgia

Orban and Ritchey (1945), on the basis of 250 cases of toothache of decompression origin, recommend that the following diagnostic procedures be employed to assess aerodontalgia.

Case History.—The following information should be obtained from the patient regarding history and circumstances surrounding occurrence of the problem.

1. Previous unprovoked pain, especially at night.
2. Previous hypersensitivity to heat, cold, sweets, or pressure.
3. Approximate age of restorations in suspected area.
4. Unusual pain during the operative treatment or during the following night.
5. Pain during ascent or descent. If on descent, it should include the length of time spent at 38,000 feet, if the problem occurred during a chamber run.
6. Altitude at which pain occurred. If it occurred at 38,000 feet, it should include the length of time the patient remained at this level.
7. Character of pain: sharp or dull, single or intermittent, intense or mild.
8. Altitude at which pain was relieved.
9. If pain persisted after reaching ground level, the length of time it persisted and whether it was followed by swelling in adjacent areas.
10. Tooth or teeth suspected by the patient.

Roentgenograms of the suspected area or side should be available.

Clinical Examination.—The following guidelines should form the framework for diagnosing suspected cases of aerodontalgia.

1. An estimate of the age of restorations in suspected area.
2. Percussion or pressure test from the lingual and buccal aspect as well as vertically.
3. Exploration for carious lesions or defective restorations.
4. Irritation with ice, heat, and electricity.
5. Removal of restorations and curettement of carious lesions.

6. Repeated decompressions tests, if necessary.

An examination of this character should give information which is interpreted according to the following guidelines:

1. Unprovoked pain, especially at night, would be a strong indication of pulp involvement, probably of an acute type.

2. A tooth that has shown previous hypersensitivity should be carefully examined. Sensitivity to heat and not to cold, or relief of pain by cold, would suggest a tooth with a nonvital pulp. Equal response to heat and cold indicates hypersensitivity (pulpitis). Prolonged pain from cold points to pulp inflammation. Hypersensitivity to sweets tends to suggest caries or a defective restoration. Pain on pressure indicates periapical irritation, but not necessarily abscess formation. (Occlusal trauma should be eliminated.)

3. Amalgam restorations of recent date are more to be suspected than are old ones or other types of restorations.

4. Unusual pain during treatment or during the night after the tooth was restored suggests pulp exposure.

5. Whether the pain was encountered during ascent or descent in the decompression chamber would differentiate between teeth with vital pulps and teeth with nonvital pulps. Pain during ascent indicates a tooth with a vital pulp; pain during descent, a tooth with a nonvital pulp, except that there appears to be a "lag" between the irritations and the responses, and thus a vital tooth might hurt briefly during descent if a very short time had been spent at maximum altitude.

6. The altitude at which pain occurs during ascent should give some indication as to the type of inflammatory process in the pulp. The lower the pain level, the more acute the inflammation.

7. Pain at 38,000 feet, and particularly after some time at this level, should focus attention on a tooth with a nonvital pulp.

8. Sharp pain during ascent would indicate a tooth with a vital pulp and with a rather acute inflammation; whereas dull pain during ascent would point to a chronically inflamed pulp. Dull pain during descent would indicate a tooth with a nonvital pulp or an inflamed maxillary sinus.

9. Very intense pain is sometimes associated with bubble formation in the pulp, even in the case of mild pulp reaction.

10. The sooner the pain is relieved during descent, the more acute the inflammation is apt to be.

11. Pain that persists for a long period after ground level is reached is

sometimes associated with teeth with necrotic or nonvital pulps. In cases of necrotic pulp, a phlegmonous swelling may develop within 24 hours.

12. Usually, the patient has no difficulty in pointing out the quadrant in which the pain occurred, but his selection of the tooth within the quadrant has been found to be unreliable. It is not unusual to have pain referred to the region of another tooth, especially the mandibular bicuspids, even when maxillary teeth are involved.

13. Roentgenograms are important in diagnosis, but they cannot be expected to disclose pulp changes or minute exposures of the pulp.

14. Observation should indicate the approximate age of restorations. Amalgam restorations of recent date can usually be recognized.

15. Percussion and pressure tests are indicative of periapical irritation, but even considerable sensitiveness is no indication for removal.

16. Carious lesions and defective restorations should be explored. The more open a lesion and the more defective the margins of a restoration, the less should a tooth be suspected of having been painful during decompression.

17. The best diagnostic aid has been the response to stimulation with ice. Nearly all teeth with vital pulps will respond to this test, but it is necessary to differentiate between hypersensitive teeth and those reacting with prolonged pain to irritation with ice. In hypersensitive cases, the response is immediate, but recovery is very rapid when the ice is removed. The expression "prolonged pain" signifies slow recovery and lingering pain. The initial response to the ice may be rapid or slow. This test should always be carried out by comparing adjacent and corresponding teeth. Only because of the relative difference in reaction is this useful. Failure of one tooth to respond to ice should suggest a nonvital pulp. It has been frequently observed that patients attempt to cover teeth with the tips of their tongues in the case of prolonged pain from ice. (Ice or ethylchloride on cotton can be used.)

18. Extreme sensitiveness to application of heat would indicate pulp degeneration.

19. The final clinical test is the removal of suspected restorations and curettement of carious lesions, with particular attention to exposed pulp horns. Exposures do not always show on inspection, but use of a sharp explorer, with some pressure, will usually disclose the concealed exposure area. Staining the lesion with an aqueous iodine solution or with silver nitrate will often mark the exposed area as a small unstained spot. All caries must be removed.

20. Repeated decompression tests will confirm a doubtful diagnosis.

Diagnostic Procedures for Medical-Dental Problems

Maxillary aerosinusitis and aerotitis media are the most common medical-dental problems causing referred pain to the teeth and thereby creating a need for differential diagnosis. While the symptoms for each ailment are varied, some generalizations can be made to assist in distinguishing dental from medical ailments.

Aerotitis Media.—The occurrence of aerotitis media may cause referred pain in the teeth and jaws. Diagnosis as aerotitis media rather than aerodontalgia can usually be made from the history.

Early dental investigators sought to explain some cases of aerotitis media in terms of temporomandibular joint dysfunction. However, it seems that there is little evidence to verify this theory. For example, the Royal Air Force subjected 1,000 men to decompression tests. It was found that only seven men consistently experienced aerotitis media symptoms during each of three decompressions. The otolaryngologic history of four of these seven cases showed contributing factors. Of the remaining three cases, only one *may* have included malocclusion or mandibular malposition as a contributing factor.

Aerosinusitis.—Kennon and Osborn (1944) indicate that referred dental pain from aerosinusitis usually occurs in the maxillary bicuspids or second molars. Most often such pain occurs during descent and only occasionally does it continue to ground level. Some patients complain of a burning or tingling sensation at the mucobuccal fold. Headaches over the eyes, numbness, pain in the maxilla, or a sensation of elongated teeth have occasionally been reported. Histories of nasal congestion, varying degrees of nasopharyngitis, or sinusitis, are often found but patients may feel that the dental symptoms are unrelated. Pulp infection can cause similar pains (after returning to ground level), but pulp tests or roentenograms can be used to distinguish this ailment from aerosinusitis.

A roentgenographic and transillumination examination of the area will usually reveal a large sinus with protuberances on the floor, occupied by the apexes of the roots of the offending teeth. Negative roentgenographic findings but sensitivity to percussion of one or more bicuspids or molars with no restorations is a strong indication that maxillary pain experienced in flight is a result of aerosinusitis.

DENTAL RECORDS AND TERMINOLOGY

The Flight Surgeon should possess a working knowledge of human dentition, terminology, and records, as a means of facilitating his interaction with Dental Officers. Such interactions occur most frequently in disorders requiring differential diagnosis and in accident investigations involving the identification of deceased persons through human dentition. Chapter VI, Articles 107 through 121, of the *Manual of the Medical Department* describes the terminology and recording techniques for accomplishing dental records. The following text is a summary of this reference.

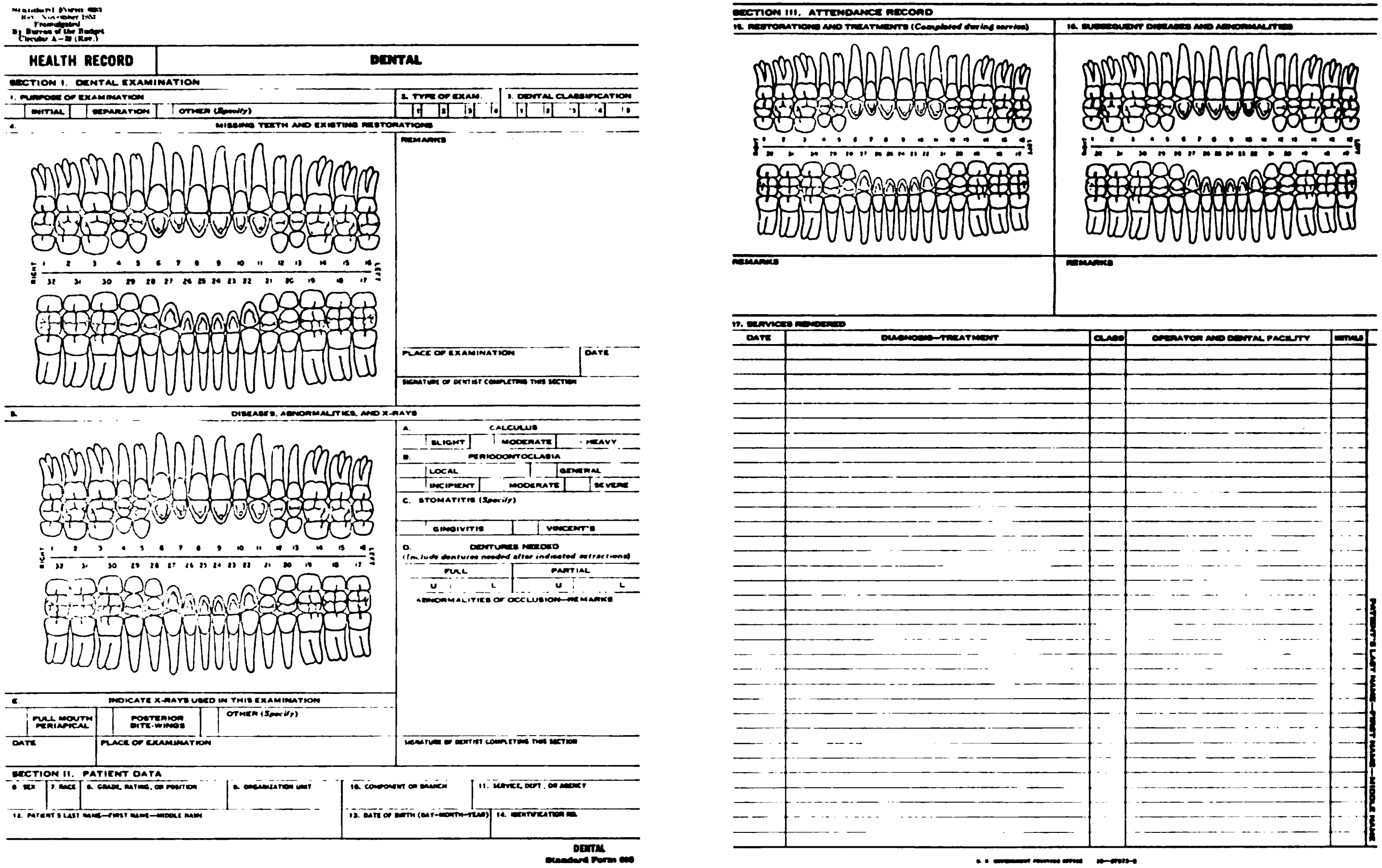
Designation of Teeth

Normal dentition consists of 32 teeth, which are numerically designated by type and position. Figure 5-1 shows the method of tooth designation used in the Navy. Standard Form 603 is the dental portion of the Health Record. Item 4 of Section I of this form utilizes this method of tooth designation as a means of recording missing teeth and existing restorations. The remainder of this discussion summarizes the most commonly encountered dental characteristics and the means employed to record them.

The surface of a specific tooth is defined as one of five possible aspects: occlusal (incisal for anterior teeth), facial, mesial, lingual, and distal. Figure 5-2 depicts these aspects for anterior and posterior teeth. These aspects are used individually or in combination to identify the position of caries, restorations, or dentures. Abbreviations for these tooth surface terms are as follows:

Surface	*Designation*
Facial (labial and buccal)	F
Lingual	L
Occlusal	O
Mesial	M
Distal	D
Incisal	I

Examples of how these terms would be used in combinations are: 22-DF, the facial and distal aspects of the left mandibular cuspid; 30-MODF, the mesial, occlusal, distal, and facial aspects of a right mandibular first molar.



RIGHT
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16
32 31 30 29 28 27 26 25 24 23 22 21 20 19 18 17
LEFT

HEALTH RECORD — DENTAL

SECTION I. DENTAL EXAMINATION

1. PURPOSE OF EXAMINATION: ☐ INITIAL ☐ SEPARATION ☐ OTHER (*Specify*)

2. TYPE OF EXAM.: ☐ 1 ☐ 2 ☐ 3 ☐ 4

3. DENTAL CLASSIFICATION: ☐ 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5

4. MISSING TEETH AND EXISTING RESTORATIONS

REMARKS

PLACE OF EXAMINATION

DATE

SIGNATURE OF DENTIST COMPLETING THIS SECTION

5. DISEASES, ABNORMALITIES, AND X-RAYS

A. CALCULUS: ☐ SLIGHT ☐ MODERATE ☐ HEAVY

B. PERIODONTOCLASIA: ☐ LOCAL ☐ GENERAL; ☐ INCIPIENT ☐ MODERATE ☐ SEVERE

C. STOMATITIS (*Specify*): ☐ GINGIVITIS ☐ VINCENT'S

D. DENTURES NEEDED (*Include dentures needed after indicated extractions*)

FULL: U L

PARTIAL: U L

ABNORMALITIES OF OCCLUSION—REMARKS

E. INDICATE X-RAYS USED IN THIS EXAMINATION: ☐ FULL MOUTH PERIAPICAL ☐ POSTERIOR BITE-WINGS ☐ OTHER (*Specify*)

DATE

PLACE OF EXAMINATION

SIGNATURE OF DENTIST COMPLETING THIS SECTION

SECTION II. PATIENT DATA

6. SEX

7. RACE

8. GRADE, RATING, OR POSITION

9. ORGANIZATION UNIT

10. COMPONENT OR BRANCH

11. SERVICE, DEPT., OR AGENCY

12. PATIENT'S LAST NAME—FIRST NAME—MIDDLE NAME

13. DATE OF BIRTH (DAY—MONTH—YEAR)

14. IDENTIFICATION NO.

DENTAL
Standard Form 603

SECTION III. ATTENDANCE RECORD

15. RESTORATIONS AND TREATMENTS (*Completed during service*)

REMARKS

16. SUBSEQUENT DISEASES AND ABNORMALITIES

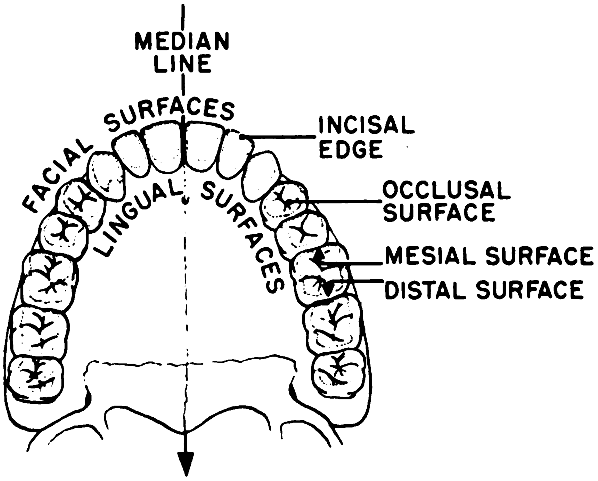
REMARKS

17. SERVICES RENDERED

DATE	DIAGNOSIS—TREATMENT	CLASS	OPERATOR AND DENTAL FACILITY	INITIALS

PATIENT'S LAST NAME—FIRST NAME—MIDDLE NAME

FIGURE 5-1. *U.S. Navy method of tooth designation.*



Median line and surfaces of teeth.

DEFINITIONS

The MEDIAN LINE is an imaginary perpendicular line that passes between the central incisors in each arch.

The surfaces of a crown are named as follows:

1. FACIAL—the surface facing the lips or the cheeks.
2. LINGUAL—the surface facing the tongue or the hard palate.
3. OCCLUSAL—the grinding surface of a posterior tooth.
4. INCISAL—the cutting edge or surface of an anterior tooth.
5. MESIAL—the surface facing the median line.
6. DISTAL—the surface facing away from the median line.

FIGURE 5-1. *Surface designations for anterior and posterior teeth.*

Restorations

Dental restorations are made from several types of materials. Each type is briefly described below including the technique for record identification.

Restorative Materials.—Three materials commonly used to restore carious teeth are as follows:

Silver Amalgam. This alloy restoration presents a silver or black appearance. The dental record indicates these restorations by a solid black mark of the same size, shape, and location as the restoration.

Nonmetallic. Nonmetallic restorative materials most frequently used are tooth-colored silicates, porcelains, or plastics. Recording of these restorations is accomplished by outlining size, shape, and location of the restorations on the dental chart.

Gold. The dental record indicates gold restorations by horizontal, parallel lines within the outline of the restoration.

Missing and Replaced Teeth

Missing teeth are indicated on the dental record by placing a large X on the root or roots of each tooth that is not visible. Common replacements for missing teeth are complete dentures, removable partial dentures, or fixed partial dentures. The recording techniques used to indicate replaced teeth are:

Removable Prostheses.—A line is placed over the numbers of the teeth replaced and the prosthesis is described briefly in the *Remarks* space.

Fixed Partial Dentures.—Each aspect of a fixed partial denture is outlined, showing size, location, shape, and teeth involved. The symbols for materials are the same as for restorations except that gold is shown by diagonal rather than horizontal lines.

Crowns.—Crowns are used to restore lost tooth structure. The material for crowns is usually gold or porcelain. The method for recording crowns (not involved with fixed prostheses) is to outline the size, shape, and location on the affected tooth.

Abbreviations

Authorized abbreviations used in dental records are presented below to assist the Flight Surgeon in his interaction with Dental Officers.

Operation, Condition, or Treatment	*Abbreviation*
Abrasion	Abr.
Abscess	Abs.
Acrylic	Acr.
Adjust(ed)(ment)	Adj.
Alveolectomy	Alvy.
Amalgam	Am.
Anesthesia	Anes.
Apicoectomy	Apcy.

Operation, Condition, or Treatment	*Abbreviation*
Base	B.
Bridge (fixed, unless noted)	Br.
Calculus	Cal.
Caries	Car.
Cement	Cem.
Crown	Cr.
Deciduous	Dec.
Defective	Def.
Denture (complete, unless noted)	Dtr.
Drain	Drn.
Dressing	Drs.
Equilibrate	Equil.
Eugenol	Eug.
Examination	Exam.
Extract(ed) (uncomplicated unless noted)	Ext.
Filling(s)/Restoration(s)	Fil.
Fluorine	Fl.
Fracture(s)	Frac.
General	Gen.
Gingival (itis) (state type in parentheses)	Ging.
Gutta percha	G.P.
Impacted(ion)	Imp.
Impression	Impr.
Incised	Inc.
Inlay	Inl.
Inserted(ion)	Ins.
Mandibular	Man.
Maxillary	Max.
Parietal	Par.
Partial	Pr.
Periapical	Per.
Pericoronitis	Pecor.
Periodontoclasia	Pedon
Porcelain	Porc.
Postoperative treatment	P.O.T.
Prepared(ation)	Prep.
Prophylaxis	Pro.
Reappoint(ment)	Reapt.
Recement(ed)	Recem.
Reconstruct(ed)	Rct.
Reduce(d)	Red.
Regional	Reg.
Reline	Rel.

Operation, Condition, or Treatment	*Abbreviation*
Repaired	Rpd.
Root canal	R.C.
Scaled(ing)	Scl.
Sedative(ation)	Sed.
Sequestrum	Seq.
Silicate	Sil.
Silver Nitrate	$AgNO_3$
Silver points	Ag Pts.
Stomatitis	Stom.
Surgical	Surg.
Suture(s)(d)	Su.
Treatment(ed)	Tr.
Unerupted	Uner.
Vincents	Vin.
Zinc chloride	Zn. Cl.
Zinc oxide	Zn. O.

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CHAPTER 6

Aerospace Psychiatry and Neurology

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DIVISION OF PSYCHIATRY AND NEUROLOGY
NAVAL AEROSPACE MEDICAL INSTITUTE

Psychology and Psychopathology of Flight
- The Evaluation of Flying Motivation
- The Selection Interview for Flight Training
- Stages of an Aviator's Life
- Fear of Flying
- Psychological Defenses Against Fear of Flying
- Disguised Fear of Flying
- Treatment
- References

GROUP PHENOMENA
- Group Structure
- Morale
- References

PSYCHOPHYSIOLOGIC ASPECTS OF FLIGHT
- Adaptation to the Everyday Environment
- Adaptation and Orientation to the Aerospace Environment
- Internal Factors Affecting the Aerospace Adaptation
- External Factors Affecting Adaptation to the Environment
- References

PSYCHOLOGIC ASPECTS OF SEA SURVIVAL
- Presurvival Training
- Aggression and Depression in the Survival Situation
- Group Situations in Survival
- Summary
- References

AEROSPACE NEUROLOGY
  Epilepsy
  Syncope
  Headache
  Head Injury
  Vertigo
  Summary
APPENDIX. "The Many Faces of Airsickness"

THIS SECTION presents the essentials of aerospace psychiatry and neurology. The material is designed and selected for either the nonaviation medical officer who wishes to obtain introductory material to, and an overview of, the field, or for reference and review by the Flight Surgeon assigned to operational units. Basic psychology, psychiatry, and neurology will not be covered, for it is assumed that all naval medical officers have been adequately grounded in these areas. For those who would like a review of personality development, chapters 1, 3, 5, 7, and 10 of the English and Pearson text (1963), and the chapter entitled *Growth and Crises of the Healthy Personality* in Erikson's monograph (1959) are highly recommended. For a brief review of descriptive psychiatry, the *Introductory* and *Definition of Terms* sections of the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders* (1952) is unsurpassed.

An article on motion sickness is included in the Appendix, organized and worded so as to make it appropriate for presentation to an audience of aviators.

The clinical material on which this section is based was hundreds of cases studied in the Division of Psychiatry and Neurology at the Naval Aerospace Medical Institute after referral from Flight Surgeons in the field. The viewpoints expressed are those of the current staff.

PSYCHOLOGY AND PSYCHOPATHOLOGY OF FLIGHT

Military aviators in general—and particularly those dedicated individuals who make up the Air Wings of our attack carriers—are men of extremely high caliber who are capable of demonstrating the finest in courage and responsibility. They are adventurous, courageous—the "cream of the crop" in many ways—and probably psychologically better motivated for their work than most other occupational groups. The first well-organized theory of the psychodynamics of flying was presented by Bond (1952) in his classic study of World War II flyers. Bond stresses the unconscious libidinal and aggressive factors in the love of flying and the symbolic meaning of the aircraft. His reader almost gets the impression that the choice of flying is largely a symptomatic act and that pilots as a group are highly neurotic. But the aviator has changed since World War II, when psychopathic qualities were so common in the fighter pilots Bond writes about.

Today's aviator is more standardized than before, computerized in his selection and training, and safety conscious. He flies a complex "weapons platform," which has no place for the "flying nut" of the past. While it is unquestionably true that subconscious forces contribute to the choice of flying as a profession, this is probably true for all choices that are made, particularly during the early years of life. All men carry forward residues from childhood, but military aviators are characterized by stability, courage, adventurousness, and a relatively unwavering trend toward maturity as time passes. Rather than ascribe their wish to fly to a specific subconscious conflict, it seems more correct to say that pilots fly because there are aircraft to be flown. The challenges such men meet may, at the same time, serve to provoke regressions, and provide the clinical material of aerospace psychiatry, but these would have occurred much sooner in lesser men.

The Evaluation of Flying Motivation

The possibility of an aircraft accident exists for all who fly. The attitude of the flyer toward this possibility, his manner of handling his reaction to it, and the factors which cause him to fly in spite of it, join or balance each other to produce his flying motivation. In evaluating flying motivation, four basic areas are important.

Origins of Interest.—We commonly find that interest in flying dates back to childhood and may reflect a carrying forward of family tradition. Thus it appears that when a pilot is firmly identified with a pilot father, his chances of success as an aviator are good—a fact which is not surprising in view of our knowledge that a successful resolution of the Oedipus complex bodes well for a successful life adjustment. These persons say they have wanted to fly as long as they can remember.

Satisfaction in Flying.—Even in the absence of a lifelong interest in flying, great enjoyment from the experience of flying itself can cause a strong identification with flying. "I'm a little depressed when I'm away from flying.

When I take off and cruise along there through the air, I leave my worries behind." Conversely, regardless of how long-standing the interest in flying, it must be enjoyable for the individual to continue. A man who wants to *be* a pilot, but who doesn't like to *fly,* will have trouble continuing. Such a flight student was anxious and ill at ease during his first military instructional flight. "I kept hoping it would go away with more experience, because I want to fly so badly. I transitioned to the T-28 and it didn't help; neither did instruments or gunnery. Finally, when I still felt that way after carrier qualifications, I decided I couldn't go it any more."

Flying Experiences.—An aviator who has had more than his share of combat or crashes will bear psychological scars. Psychological problems will undoubtedly arise, as well, for the aviator ordered to replacement training as a jet fighter or attack pilot following six or so years of minimal and proficiency flying.

Reactions to Accidents.—Most naval aviators have been exposed to fatal accidents among their immediate acquaintances. How do they cope with this reminder that they may be next? Rationalization is often effective. "People get killed everywhere. I'm in greater danger driving my car." (In truth, more flight students *are* lost in automobile accidents than in aircraft accidents.) Healthy denial is reinforced by attributing the accident to some faulty act of commission or omission which other aviators always avoid and would never do. "He was a good pilot, but . . . ." Less healthy reactions are, on the one hand, a panic which grounds the aviator for over 24 hours, and on the other hand, a complete repression of the whole topic. "I have no reaction to crashes."

The Selection Interview for Flight Training

Each applicant for naval flight training must be interviewed by a Naval Flight Surgeon, and each interview requires at least 45 minutes. This is the irreducible minimum, and any examination done in less time should not be termed a selection interview for flight training. Safe ground on which to start this interview is to ask about the candidate's prior flying experience. The individual's probable tolerance to high speed, turbulence, risks, and taking chances can thus at least be estimated. In an interview as brief as this, it is seldom productive to explore sexual matters. It may be simply sufficient to inquire of the applicant's marital plans, and if the applicant expresses any interest or desire to pursue this subject further, the examiner can follow his lead. Religious viewpoints and the amount of energy the applicant expends in religious pursuits will give some understanding of the individual, especially in regard to the flexibility of his personality and his ability to appreciate viewpoints other than his own. The examiner can make a judgment as to whether the religion is utilized in a healthy or in a defensive fashion. A healthy search for meaning is one thing; a constricting reaction formation is another. The applicant's educational goals to date should be explored. Long-time occupational goals which have little in common with the duties of a military aviator should at least arouse the examiner's curiosity.

The Flight Surgeon can also be guided in appraising the candidate's likelihood for successful completion of training by some of the candidate's previous life experiences. For instance, the candidate's chances are enchanced if his father was a flyer or he has had a close personal friend who has been a licensed aviator. Here, of course, one is looking for identification with aviation. The candidate's chances are improved if he has had some flight instruction himself, and particularly if he has soloed. This is an example in itself of strong motivation. It's a better candidate who knows the basic language of aviation (words such as *skids, slips, mushing,* and *stalls*). Such knowledge indicates both motivation and identification. The candidate will probably do well who thinks he is in better physical condition than most of the other applicants, and that his physical reactions are faster than those of most flight trainees. These things indicate a healthy self-esteem. The candidate who has had mechanical or manual hobbies has demonstrated both his manual dexterity and the general area of his interests. Probably the two best motives for applying for flight training are the candidate's feeling that he has an aptitude for flying and that he is doing it for career purposes.

It is reasonable to suppose that any healthy, red-blooded, interested American boy of normal intelligence and social skills should be able to learn to fly the Navy's aircraft competently.

Stages of an Aviator's Life

Some generalizations regarding the early lives of aviators-to-be seem warranted by clinical experience. It is helpful to remember that fear of flying (in an abstract sense) is one of the earliest sensations known to man—as exemplified by the startle reaction in an infant who is suddenly dropped. This inherent fear of an unstable, unsupporting environment persists, and must be dealt with by counterforces, suppression, repression, or limiting the scope of one's life. There are some who go through life fearfully. For those who cope with it successfully, the fear is converted into thrill. *Thrill* may be considered as fear denied or rationalized. On a roller coaster, or while doing a loop, one may feel, "See! *I'm* not afraid. I'm going to be all right!"

For those who make a career of flying, four psychological periods can be distinguished, and the sequence is so predictable that it can be accurately referred to as a maturational process.

The Glamorous Years.—In the first two to four years of flying (usually to about age 24), there are few inhibitions resulting from age or experience. Much personal satisfaction derives from the mastery of a powerful and potentially dangerous instrument. Our observations strongly suggest that the personalities of successful military aviators are characterized by a great "mastery drive." They fly dangerous aircraft in a dangerous manner because the aircraft and missions are there.

Increasing Caution.—In the next four years (to approximately age 28), the pilot has become aware of his limitations and those of his aircraft. He has had narrow escapes, may have had an accident or ejection, and has lost friends who were admittedly as competent as he.

Controlled Fear of Flying.—Over the next ten years, increasing conservatism balances any tendency toward recklessness which may have existed at earlier periods, and the pilot is at the peak of perfection. He knows he can be killed, tries to avoid it, and takes great pride in being a true professional. The developing caution of this period may be considered as preparation for the aging of the pilot's physical and physiological capabilities.

The Safe Years.—From about age 38 on, the pilot's great experience, combined with the conservatism of his years, make his flying the safest of his career.

Fear of Flying

Acute Anxiety Associated with Flying.—In people with such a "traumatic neurosis," the major source of anxiety is a single situational episode associated with moments of stark terror and other unusual affective states. A crash, even though survived, is a strong attack on the aviator's myth of his own invulnerability. In such cases springing from a single episode, it is generally true that "the greater the stress, the better the prognosis." The prognosis for return to flying, particularly with superficial psychotherapy and support by the Flight Surgeon, is good.

Chronic Anxiety Associated with Flying.—Chronic anxiety has an insidious onset and is not related to any specific episode. Rather, there are multiple sources of origin, including subconscious forces triggered by the act of flight and the responsibilities of being a pilot. The "straw that breaks the camel's back" may be family problems, predisposition to neurotic breakdown, loss of enjoyment of flying with advancing age, too many frightening incidents through the years, or requirements to learn new aircraft and techniques. Sometimes a phobic reaction develops, in which, through use of the mechanisms of isolation, condensation, symbolization and rationalization, the multiple or "nameless" fears are focused on one (often realistically minor) aspect of flying. This process enables the aviator to say to himself (inaccurately), "If I can just avoid turbulence/great altitude/the F-8 aircraft, everything will be fine." Since the avoided state is not the real object of the fear, a "spread" of the symptoms to other areas of flying is the usual course. A 38-year-old aviator illustrates this type of problem. He was married to a woman nine years older, who was chronically ill with phlebitis and back trouble. He had requested straight squadron tours throughout his career, which one suspects of representing a counterphobic or overcompensation mechanism. He had been shot down in Korea, had ejected twice (once from an aircraft in flames), and had experienced oxygen equipment failure leading to anoxia on one high-altitude flight. When ordered to replacement training as prospective commanding officer of a fighter squadron, he was not able to fly off the ship at night for fear that bad weather might require a climb to great altitude. His fear of altitudes over 30,000 feet persisted, and he had to be relieved of his command.

Psychological Defenses Against Fear of Flying

When we are young, we think not only ourselves, but that all about us, are immortal.—DISRAELI.

An aviator may use conscious mechanisms to lessen his flying anxiety. He may request helicopter training rather than jets, or orders to a multiengine shore-based squadron, or arrange for nonflying squadron duties to take up most of his time. He may become adept at finding excuses to avoid scheduled flights. He may increase his confidence in the aircraft by learning a great deal more about it than is required, and by exhaustive preflight inspections. A thorough knowledge of emergency procedures may serve the same purpose—a quick answer at his fingertips (he hopes) for every conceivable inflight problem. Or he may consciously and deliberately decide to "stay with it," refusing to quit, no matter what the cost in mental anguish. Suppression is often used to modulate the memory of a panic state.

Of the less conscious defenses used, by far the most important is *denial.* Denial, with its associated feelings of omnipotence and invulnerability, gives rise to such thoughts as, "It's not *really* that dangerous," or "It will happen to the next guy; it can't happen to me." While the minimal use of denial is essential if hazardous activity is to be continued, its excessive use strains the forces of repression to the breaking point and symptoms appear.

Counterphobic techniques sometimes enable the pilot to "fight away" his anxiety. A flight student with intense unilateral headaches associated with flying was, nevertheless, forcing himself to "stay with it." He requested always to be first on the day's flight schedule to get it over with, a counterphobic device.

Identification takes place with admired groups, both large and small. An example of the former is all of those who wear the Navy "Wings of Gold." An example of the latter is an admired, close, and effective jet fighter or attack squadron which takes great pride in being a closed-membership group with high admission requirements. Through this identification, strength is borrowed, and weakness is for a time forgotten.

Disguised Fear of Flying

If an aviator is assigned flying duties which are too anxiety provoking for him to bear, several possibilities are open to him. He may simply turn in his wings, requesting assignment to nonflying duties. If he does this, however, his friends may think him "chicken" and, even worse, a wide and unacceptable breach would be created between his self-concept and his ego ideal; or he may perform so poorly that his aviator's status will be taken from him. This may be very dangerous; and, again, may be too much of a blow to a precarious self-esteem. The alternative which many seek is medical grounding. "Seek" here refers to primarily subconscious determinants. Removal from flying because of "organic" symptoms causes no conflict between self-concept and ego ideal. On the contrary, an aviator who is protected from flying by such symptoms can afford to declaim his motivation more loudly than usual—and that's the beauty of this alternative. Apparent physical illness is an acceptable cause for grounding in any social or professional circle; fear never is. A man so grounded will encounter compassion from others, pity from himself. There is little wonder that pseudo-organic symptoms of psychogenic origin are so frequently encountered.

To establish a symptom as a disguised fear of flying, several criteria must be met. First, it is noted that denial is used not only for hiding the specific fear, but that it spreads over and covers aspects of flying that are, to a degree, frightening to everyone who experiences them. Transitioning to a new high-performance aircraft is never accomplished without moments of great uneasiness, and the healthy pilot can discuss this. Shipboard jet operations at night are dangerous for all, and the aviator not torn by inner conflicts can openly acknowledge that this is so. The "I'm not afraid of anything; I'm just sick" attitude is easily detected in an interview.

A second criterion is that the symptom should be of such a nature that it would, in the aviator's estimation, disqualify him from a certain type of flying, but from little else. Here, the aviator's medical and physiology training has taught him that anything impairing spatial orientation (vertigo), vision (inexplicable blurring or defective but highly variable visual acuity), or consciousness (even for the briefest conceivable moment) is of great significance in flying. Illustrative of this knowledge is the aphorism, "Eyes are to the aviator what legs are to the infantryman."

Third, in spite of a strongly stated desire to fly, the patient has actually stopped flying because of the symptom. It is often helpful to realize that many other aviators have not curtailed their flying, even in the presence of organic problems of greater magnitude.

The fourth criterion is that the aviator must be in a new or approaching situation of greater flying danger. There would be no reason for an aviator to use such symptoms, for example, at the start of a tour during which he would do only proficiency flying with minimum hours. There must be something relatively new and very threatening. A common seedbed for such symptoms is a Carrier Replacement Air Wing, where second- and third-tour pilots have to re-night-qualify in new aircraft with new techniques after one or more tours "flying a large mahogany desk."

The disguised representations of fear of flying can be grouped into conversion reactions, hypochondriasis, and psychophysiologic reactions. *Conversion reactions* in aviators are quite subtle and always involve senses very obviously required to fly. A 28-year-old aviator, three months after a crash into the water and just prior to a carrier deployment, reported several instances of feeling himself to be upside down for many seconds while flying at night at sea. This disorientation obviously prevented his going on the cruise.

Hypochondriasis is frequent, and may be defined as an obsessive overconcern with the state of one's health. Attention is shifted from the threatening environment and frightening reality situation to the pleasanter contemplation of minor variations in anatomy, normal physiological functions, and minor ills.

Psychophysiologic reactions may or may not be used to achieve the secondary gains associated with removal from dangerous flying. The diagnostic criteria given earlier are applicable only to the extent that such reactions are used for the purpose of changing a threatening environment. The key word for psychophysiologic reactions associated with flying is ***ambivalence,*** for here a strong motivation for flying is combined with a deeply buried and extremely great fear. It should be remembered that one of the characteristics of the subconscious is that strong opposite feelings and ideas can continue to coexist face to face, but not peacefully; psychophysiologic reactions involving the autonomic nervous system often represent a sort of cold war. To many of these people, the symptoms are troublesome but are deliberately borne or ignored so that the joys of flying can be theirs. They cannot be trusted to evaluate the hazard to flying that their symptoms present. Removal from flying in these cases should depend simply on how disabling the symptoms are.

A young Coast Guard flight student was very uneasy during his first three airplane flights. He then lost his apprehension, but simultaneously developed headaches associated with a labile blood pressure. When these were interpreted to him, and he was told if they continued he would be dropped from flight training, they disappeared, only to be replaced by nausea, abdominal cramps, diarrhea, fatigue, and impotence. Returned to flight training once more, after a tearful appeal to be allowed to do so, he became stricken with anxiety prior to his next hop, and to avoid the flight, blurted out that he wished to be dropped from the program. This man's fear of flying progressed from full, conscious awareness to an exaggerated sympathetic response, then shifted to parasympathetic overactivity, and finally emerged again as conscious fear.

Treatment

In acute reactions to severe trauma, catharsis (the verbal kind), rest, and a prompt return to flying in gradually increased increments of responsibility are indicated. Sometimes the type of mission assigned may have to be modified (downgraded in complexity) for a short while, but always with the goal of resumption of unrestricted flying clearly in mind. Supportive therapy of this sort suffices in most cases; rarely will expressive psychotherapy (uncovering of deeper, more primitive feelings and their correlation with the significant past) be needed.

In the less acute manifestations of fear of flying, the patient sometimes cannot accept the concept that he is afraid and that this fear is what produces the symptoms; the full awareness of such a "blemish" would overwhelm his already precarious self-esteem. In such cases, it is in the best interests of preventive psychiatry to let him blame the Flight Surgeon for his loss of flight status and for his Flight Surgeon to accept this blame with equanimity. While this involves the use of pathologic mental mechanisms by the patient, the only alternative available to him would often be a crippling depression.

A return to flying, even in the presence of considerable fear, can nevertheless be accomplished in many cases. In some instances, the installation of *hope* is valuable. (This is emphasizing that there will be a better tomorrow, that the unpleasantness will end at an easily visualized point in time.)

Motivation which is waning because of fear can also often be bolstered by an awareness of the (career and pay) losses entailed in the change to a nonflying status. At times of panic, long-range goals tend to be forgotten. In an interview with the Flight Surgeon (who should be nonjudgmental and allow the patient to develop his own thoughts), the aviator can often become aware of all he is giving up, and decide to make a greater effort.

Occasionally, the Flight Surgeon's assurance that the *feelings* are no adverse reflection on the individual and are not incompatible with later success, can do much. Finally, the Flight Surgeon's friendship and interest as the physician member of the same group, can do much to keep the indecisive aviator with the organization. The Flight Surgeon whose identification with the wing or squadron is solid can be of great influence by saying, "*I* care, and I'd like to see you stay with us. I know it's hard, but I think you can make it." Square pegs can't be forced into round holes, but the shape (personality) of the pegs (pilots) sometimes requires considerable elucidation.

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GROUP PHENOMENA

The study of groups and group behavior is one of the newer areas of psychiatric investigation. Group phenomena are particularly important to the military physician.

Group Structure

Groups, like individuals, have identities. In the military, great emphasis is placed on establishing this identity. We have devices, patches, sayings, posters—all distinguishing, visible marks of a group identity. These are attempts to establish group feeling and loyalty. We share a group identity as physicians. This identity as a physician is nowhere more apparent than to the student Flight Surgeon going through primary flight training. At this time, many students, because of initial anxiety, try to convince their flight instructors that they are not really flight students, but doctors. After a short period of time, and when the student Flight Surgeon is given control of the aircraft, he gradually releases his identity as a physician, and assumes that of a flight student.

The primary characteristic of a group is that there must be more than two people to be a group. The next most important aspect of a group is that there is a group feeling, or underlying group emotion, present in any group. This group feeling is not present at all times, however. It is most present when the entire group is under some emotional state. This state can be any emotion—anger, hostility, humor, or adulation. The well-known situation of sitting in a boring lecture is a good example. If someone makes a comment that infers the lecture is boring, there is usually a wave of head nodding, and there is a general feeling of assent throughout the class. This is almost tangible at certain times. These group feelings are particularly evident in therapy groups. However, in any group, be it a survival situation, be it a squadron, there is a detectable feeling, if you will take the time to notice it. This can be resentment at flying too many hours, or a feeling that the squadron is overworked. Although this is not verbalized, it can be observed in the way people drag into the ready room, come late to briefings, and so on.

Another important characteristic of a group is its mythology. The "Green Berets," for example, not only mark their wearers as different, but as the "cream of the crop." Along with the group mythology, there are also "group rules," written and unwritten. An example of an unwritten rule is the frequent one in some large organizations of, "Don't make waves." An unwritten rule in the Navy is that no one sits in the CO's chair in the ready room.

Just as individuals have significant interactions and processes, groups also have significant interactions and processes. A typical group interaction involves competition and rivalry between various divisions and squadrons. A "paranoid group" feels picked on and threatened by the rest of the surrounding groups and environment. In this manner, they perpetuate their isolated community. Only their members are accepted, and the rest of the group's world; and individuals who are not in this group are looked upon as outsiders. They usually have a strong group feeling. In such a group, the group *feeling* is one of paranoia; the group *mythology* is that they are persecuted by the rest of the world or a large segment thereof. A primary rule is, "Be paranoid or don't join our group."

Morale

Particularly important in the military are factors of morale. In reviewing many of the things written about morale, four basic principles are evident: faith in a common principle; faith in the leadership, faith in each other; and adequate health, with a balance of work, rest, and recreation. The first three principles are of note in that they all require of the individual the ability to form both conscious and subconscious identifications. The individuals in the group must be able to have faith in their principles, leadership, and in their colleagues. They must be able to identify with them, to empathize with them emotionally, and to interact with them effectively. Some people with character disorders, who have had difficulty throughout their lives in forming relationships and in finding anyone or anything to identify with—or, in fact, lack this capacity completely—make very poor and often difficult members of a group.

Sick Call can often serve as an indicator of the nature of the group phenomena in the area. In other words, are the patients, predominantly, from one division? Is there one person with whom many of the patients in Sick Call seem to be having difficulty?

It is interesting that "brainwashing" centers about group phenomena. One of the prime techniques involves separation of the leaders from the group. As potential leaders develop, they also are separated from the group. As time moves on, some group members begin to confess things about people close to them. Mutual confidence is shattered. There are rewards for tattling or squealing, all in the guise of, "We're just trying to help and educate." Finally, all group feeling is lost; all sense of identity is lost; the group can not organize for effective action, such as escape. A successfully "brainwashed" group is a study in negative group dynamics.

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PSYCHOPHYSIOLOGIC ASPECTS OF FLIGHT

The interplay of environment and behavior is nowhere more vividly highlighted than in aviation medicine. The aviator is continually and simultaneously responding to events that occur in his internal or bodily environment *and* the external or aerospace environment. In this section, the psychophysiologic and environmental factors that affect the efficiency of the aviator's responses are discussed. These factors are relevant to the manner of man's relations to his surroundings, and especially to *new environments,* e.g., aerospace, the sensorially deprived or sensorially excessive environment. *Internal changes* such as orientation, vertigo, and sleep deprivation also affect this relationship.

Adaptation to the Everyday Environment

All sensory inputs, visual, auditory, vestibular, pain, touch, and muscular, serve to provide orientation in space. These inputs are integrated centrally to give us a mental, though often subconscious image or impression of our body's position in space. These inputs are also affected by emotional states. When we are tired or depressed, our limbs feel heavy; when elated, we feel light and buoyant. Due to the variability of emotional states and the demands of the environment, the image we have of our bodies is constantly changing. Through trial and error and practice, our ability to move effortlessly in space is developed, and our success leads to a sense of *perceptual-motor control of the environment.*

In our daily lives, we have complete perceptual-motor control of our environment; we give little conscious thought to our actions. Through learning and habit, we automatically judge distances, walk across rooms and up stairs; unconsciously we know where our arms and legs are in relation to space. There are times, though, when these perceptual-motor skills go awry; for example, when we walk down stairs and find that we anticipated one more step than there actually was. We bring our foot down quite hard and unexpectedly and immediately wonder what happened, what went wrong—and for a brief moment, our perceptual-motor control of the environment is lost; we become disoriented in space. This experience can create anxiety, because of which we may be especially careful on stairs for a few days, particularly those same stairs. Such anxiety usually is quickly forgotten. A more vivid example of anxiety created by the loss of perceptual-motor control is when we are about to fall asleep and suddenly feel we are falling. The resulting anxiety does not completely leave until we are fully awake and reassure ourselves that we are at home, safe in our own beds. Fortunately, these experiences are rare, and we are usually comfortable with our bodies and movements through space. The anxiety from disorientation in our daily lives is qualitatively the same as that associated with disorientation in flight.

Adaptation and Orientation to the Aerospace Environment

Adaptation to the aerospace environment is a vivid, practical demonstration of the blending of perceptual-motor mechanisms and emotions. The new flight student suddenly finds himself in a very strange perceptual environment. Initially, everything is alien to the new student aviator: The plane seems to have a will of its own, the clouds are enormous, and nothing on the ground looks familiar. In the cockpit on the first few flights, the student is rigid; he seems to feel that if he moves or jostles the instruments, the aircraft will tumble from the sky. His grip on the controls is like a "death grip"; he seems to be trying to hold the plane in the air by this one control. As the student's training progresses, he begins to feel that the plane is responding to his commands; the plane is doing what he wants. And when he finds the plane responding to his commands, much as his arms and legs do when on the ground, he not only feels more comfortable, but has also incorporated the physical aspects of the plane into his own body image. We might say that he is now fused with the plane on a perceptual-motor level; he no longer thinks of where the wings are or where the landing gear is, but he knows—just as he knows on the ground where his arms and legs are in relation to objects around him. When the student finds that he can maneuver the plane at his will, and thereby himself, through the three-dimensional environment in flight, he has achieved perceptual-motor control of the environment. But to do this, he must feel that he is *fused* with the plane, i.e., that the plane is acting as a part of himself. It is interesting to note that many experienced pilots feel anxious when they are passengers in commercial airlines, and can achieve some degree of comfort only by reviewing the flight procedures the actual pilot is performing. One should also note the different feeling experienced by an aviator when flying as a passenger. The plane in this situation is no more than a vehicle, a bus. However, this feeling changes if he takes the controls. The plane then becomes something different. He then has a much more personal relationship to the plane, the boundaries and image of the plane seem to fade away as he is lost in the process of flying; it is no longer a conveyance, but is a part of himself, something he himself is doing. The physical sense of separateness from the plane seems to blur away, leaving a feeling of oneness.

As the student progresses through training, each new phase—instrument flying, formation, carrier landings—presents a new perceptual task which he must master in the same manner of perceptual-motor control and fusion. Once these tasks are mastered, little thought is given them. If the pilot is not particularly comfortable in one phase of flying, it is frequently because he has never been able adequately "to feel in control" of the situation and thereby the aircraft. The most common area of difficulty is instrument flying, and it is not uncommon to see pilots with long careers in aviation who have managed to limit their actual instrument hours to a minimum.

Internal Factors Affecting the Aerospace Adaptation

In the early stages of learning to fly, and when learning new flying skills, the student is in a continual state of disorientation. Without a sense of perceptual-motor control of the environment, he experiences what can be termed *perceptual anxiety.* Perceptual anxiety is present in the patient struggling awake from anesthesia. It lasts until he is oriented and remembers what has happened. The frequent anxiety of people with organic brain syndromes is in part related to their loss of perceptual-motor control of the environment.

The anxiety in learning a motor task is soon forgotten, once it is mastered, whether it be swimming, riding a bicycle, or flying an airplane. However, when the pilot becomes subject to the perceptual illusions associated with flying, such as false sensations of rotation, or seeing the plane banking left but feeling it bank right, the plane then again becomes something alien. Fusion with the plane is broken; the pilot's sense of control of the environment is lost, and anxiety results. Disorientation frequently leads to the breakdown of habit patterns, and further to accidents. Disorientation, "pilot's vertigo," is definitely not one of the sought-after sensations of flight. In fact, pilots tend to limit their flying to situations which do not provoke vertigo, once they have experienced it in a particular situation, e.g., night flying or instrument conditions.

Disorientation experiences can be grouped into four broad categories (Clark & Graybiel, 1955).

1. *Visual Illusions.* These include, for example, autokinesis (the apparent motion of a light source at night or in dim light); and the problems of relative motion, e.g., the feeling that objects you are approaching, such as other planes, are moving, but you are not.

2. *Nonvisual Illusions.* These include failure to perceive rotation, or the perception of rotation when there is none (oculogyric illusion and Coriolis effect); or the feeling that one is climbing or diving when one is flying straight and level but accelerating (oculogravic illusion).

3. *Conflicting Sensory Cues.* These include such feelings as that of banking to the right when the visual horizon indicates that the aircraft is flying level.

4. *Dissociational Phenomena.* These include such things as loss of time or directional sense and "fascination" with one particular aspect of flight.

All of these illusions are more common at night, in rain, fog, and haze (particularly when flying over water), and result from reduced visual references. Other internal factors are (1) the pilot's instrument proficiency, (2) his confidence in instrument flying, and (3) his physical condition, particularly in relation to fatigue, boredom, and sleep.

The effects of sleep deprivation, combined with a stressful environment, can be marked and surprising. World War II troops who were kept awake nights on end by harrassing enemy action were frequently incapacitated by psychoticlike behavior. Many college students who lose sleep during a stressful examination period experience a brief psychoticlike reaction. The term "psychoticlike" is used because, in most of these people, there is no hint of historical or personality proneness to psychosis and the reactions usually clear up after adequate sleep.

It has long been noted that a "model psychosis" can be developed with sleep deprivation (Luby, Frohman, Grisell, Lenzo, and Gottlieb, 1960). In subjects sleep-deprived anywhere from 72 to 220 hours, a wide variety of emotional symptoms (irritability, expansiveness, grandiosity, paranoid states, visual hallucinations, and rage attacks) are noted. More pertinent in the case of aviators is the fact that, even after 24 hours of sleep deprivation, changes in length of reaction times and increased errors in psychomotor performance can be noted. In the sleep-deprived person, the most common symptom is *unevenness of performance;* it is not a straight-line declination of performance. This unevenness of performance is due primarily to lapses of attention. If the subject performs tasks at his own pace (subject paced), his reaction time is simply increased; however, if the task is paced by the experimenter (time limit), there are increased errors (figures 6–1 and 6–2, Williams, Lubin, & Goodnow, 1959). The implications of these data for aviation personnel are self evident. The lack of sleep not only alters the person's internal environment with regard to psychomotor performance, but sleep deprivation may precipitate seizures by lowering the convulsive threshold (Bennett, 1963).

External Factors Affecting Adaptation to the Environment

It has long been noted that people in isolated situations—arctic camps, prisons, patients in iron lungs, and survivors at sea—have hallucinatory and delusional experiences (Solomon, Leiderman, Mendelson, & Wexler, 1957; Wexler, Mendelson, Leiderman, & Solomon, 1958). Any environment where there is *constancy of the surroundings* can provoke a sensory deprivation experience. The exact etiology of these experiences is problematic, but the reality of the psychologic and physiologic effects has been well docu-

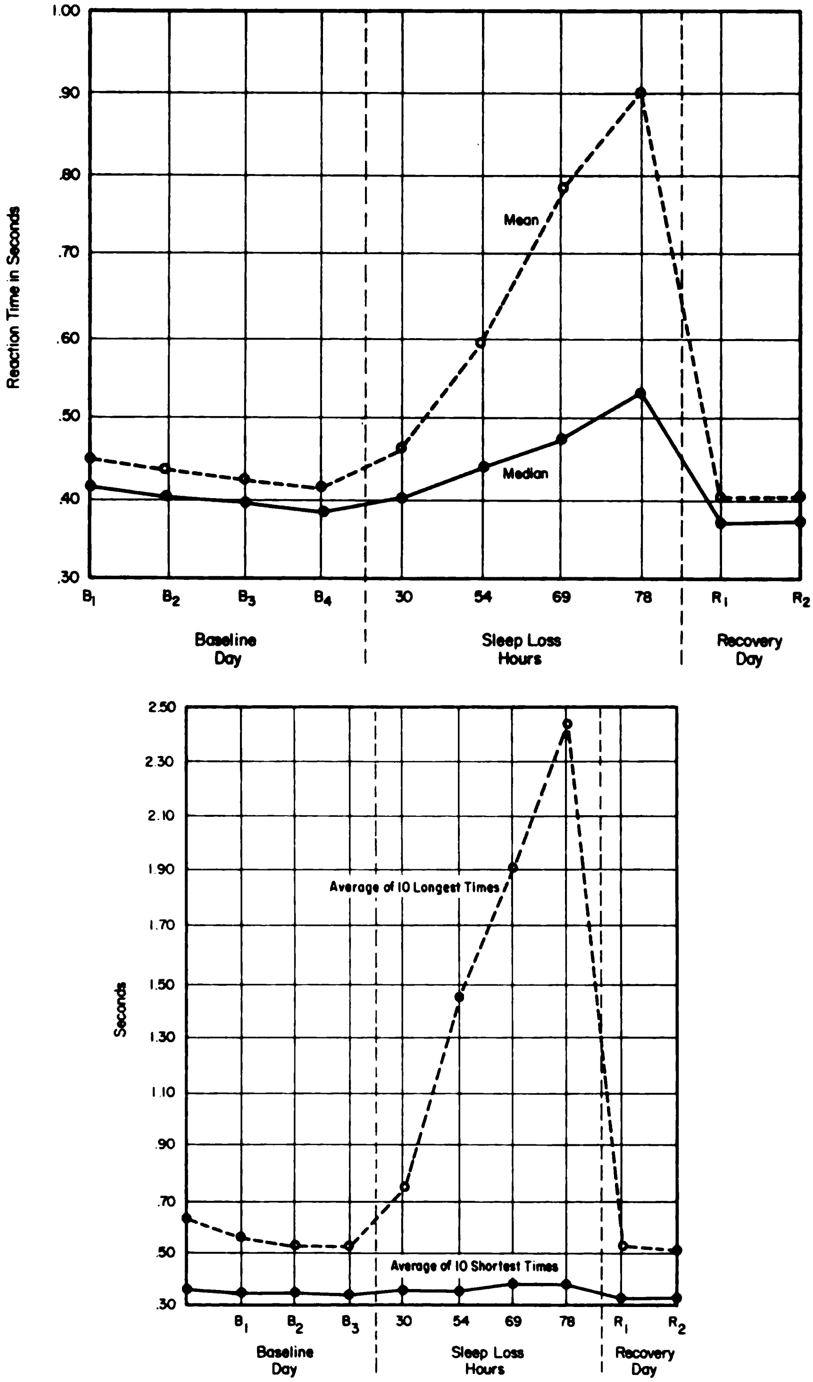

FIGURE 6–1. *The effects of sleep loss on reaction time* (WILLIAMS, LUBIN, & GOODNOW, 1959).

mented. In any situation where there is a decreased patterning of the sensory stimuli, changes of great emotional intensity and psychoticlike experiences can occur. These effects can be noted in even a few hours of sensory deprivation. The main effects are: (1) directed and organized thinking becomes difficult, (2) there is increased suggestibility, (3) a feeling of need for extrinsic stimuli and body motion becomes intense, (4) there is great time distortion, (5) in prolonged experiences, hallucinatory experiences are common. The perceptual organs also have difficulty in adjusting after the sensory deprivation period. Objects appear to swirl, and shapes and lines are distorted. A type of sensory deprivation experience in jet pilots described by Clark and Graybiel (1957) is called the "breakoff phenomenon." This is a phenomenon noted primarily when flying alone at altitudes above 30,000 feet and when there are no unusual demands on the pilot for controlling the aircraft. Some pilots experience feelings of detachment from the earth and feel that they, themselves, even without the airplane, are floating off into space. Pilots who experience this phenomenon are either very upset by it or exhilarated by it, but few, if any, go out of their way to seek it. In this instance, we might say that the pilot is no longer fused to the plane, but has transcended boundaries. Bridgeman, the X–15 pilot, describes vividly the experience of fusion and breakoff.

> Fifty-nine thousand, sixty thousand, reeling off sixty-one thousand. I have left the world. There is only the ship to identify myself with; her vibrations are my own. I feel them as intensely as those of my body. Here is a kind of unreality mixed with reality that I cannot explain to myself. I have an awareness that I have never experienced before, but it does not seem to project beyond this moment . . . and with this adrenalin-inflicted state floats the feeling of detachment.

There are other more psychologic aspects of man's relation to the aerospace environment. Psychologically speaking, there are drives to motor activity. The discharge of these motor drives may be of major motivational significance in flying. Visualize the pilot sitting in the cockpit. He moves very little; in fact, he is almost immobilized by the equipment and restraining straps, yet he is producing great motion in his aircraft. He is transferring his motor functions to the plane, and thus releasing those motor drives which must be released. His ability to fuse himself perceptually with the plane permits motor expression and may account for much of the satisfaction he derives from flying. A man who fears uninhibited motion and its emotional accompaniment can neither adapt to nor enjoy flying. The pilot's

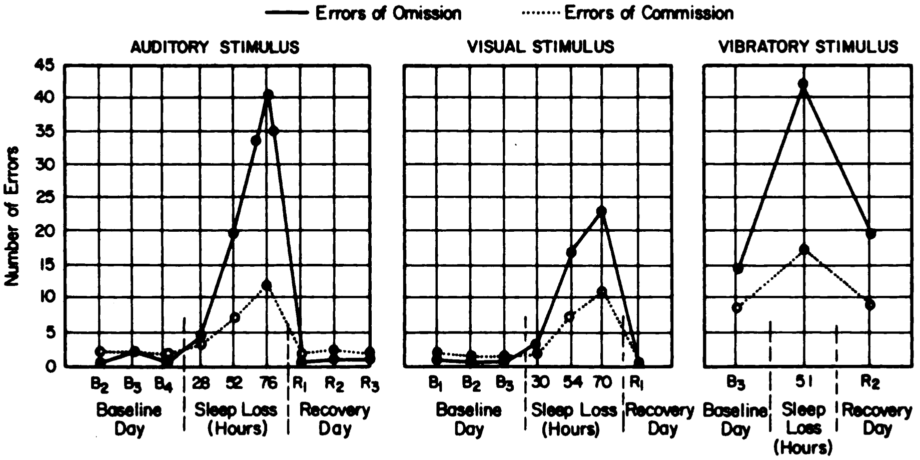

FIGURE 6–1. *The incidence of errors on three vigilance tasks* (WILLIAMS, LUBIN, & GOODNOW, 1959).

need for motor expression can be quantified. Many depressed or troubled aviators state that the only time they feel better or relaxed is when they are flying. The motor expression or release of motor tension accomplished by fusion with the aircraft is the most likely factor which provides the therapeutic or positive effect.

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PSYCHOLOGIC ASPECTS OF SEA SURVIVAL

If one closely examines the numerous case histories and personal experiences of those who have had survival experiences, one is impressed by one fact: the mental attitude, the will to survive, the determination and stubbornness of the individual are paramount. On the one hand, one is continually impressed by the fact that, despite a plethora of equipment, quantities of supplies, and physical well-being of particular persons in many survival episodes, some did not survive; and, conversely, the histories reveal that many cast to sea on pieces of wood, rafts, and with almost no equipment at all, have endured great hardships and have ultimately survived.

The act of surviving is probably one of the most individual acts which a person can undergo. Aside from the more personal and emotional aspects of survival, there is an important environmental factor in the survival situation that is a great source of anxiety and fear. This is the abrupt shift from an extremely familiar environment to a very strange and markedly different environment. The individual is dramatically faced with a loss of control of his environment; he exists at the whim of natural elements to which he had given only minor thought in the past. The successful survivor will attempt to regain some control of this environment; he will become active, he will attempt to plot a course, to obtain food, to bring some familiarity into this new environment.

Presurvival Training

It is the function of presurvival training to provide some sense of familiarity to the survival situation. In this way, the transition is not so abrupt; the environment has some familiarity. The hardware of survival has some familiarity, and one can take an active role in the survival task. As the environment can be made more familiar and control of the situation increases, panic becomes less likely. The development of the attitude that the individual *can* do something about his situation is the prime function of survival training. Although there is no clear criterion as to who will be a good survivor and who will not, the degree of familiarity the person has with the survival environment is extremely important.

In reviewing the characteristics of survivors, particularly at sea, one is initially impressed with the fact that they have made an active decision that *they are going to live*. Most of those successful at survival made some attempt to control, combat, and manage their environment. The survivor must keep busy and attempt to organize the situation along familiar lines. He must plan definite goals and strive to achieve them. He must also attempt to recall everything he has learned and heard that has anything to do with outdoor living, the sea, and surviving.

Aggression and Depression in the Survival Situation

If one is not busy and active, thoughts that had only been given fleeting notice begin to preoccupy the survivor's mind, such as, "Why did this happen to me?" He begins to search for answers, feels guilty or depressed, with feelings of hopelessness. Inertia and passivity take over. Technically, it is the problem of how the individual handles aggression. A depressed person is one who has turned his aggression inward due to preoccupation with guilt feelings. In the survival situation, the aggression must be turned outward, the struggle must be against the environment. As will be shown later, it is often the function of the leaders in a survival situation to mobilize this aggression in the form of anger at the leader. Naturally, one cannot, and should not, be in a frenzy of activity, but the activity is more of a relentless preparation, physical and mental, for survival and rescue. Every action should be directed toward this positive goal. Signalling gear should always be ready and constant attempts should be made to gather some form of food.

Group Situations in Survival

The characteristics of group survival are very similar to the individual's requirements for survival. In group survival situations, the key factor is good organization. Those survival groups which are best organized usually have good morale. The qualities of this organization depend very much on the leadership of the group. Primary to establishment of a functioning survival group are adequate briefings by those in charge or by those who have assumed leadership. The group needs to be kept busy, watches set for the sighting of rescue vessels, jobs assigned, and the survivors taught to use all signalling devices. Morale increases as the sense of purpose, activity, and interdependency in the group develops.

Summary

Many aspects of the survival situation are not covered here. There are many individual and idiosyncratic reactions. There is also the problem of the injured survivor. But basic to all survival situations, whether it be a matter of minutes or a matter of days until rescue is accomplished, there is always the initial, abrupt transition from a familiar environment to a very new and strange one. There also must be the active decision on the part of all survivors that they are going to survive. Much of this can be effected by survival training prior to the emergency. The capacity of a group for survival is directly proportional to morale. Morale is created by organization and designation of responsibilities to individuals. The leader must not allow the group to degenerate into a state of panic or apathy. Fortunately, due to the state of initial emotional shock in an emergency situation, even large groups of untrained people can be safely managed and controlled by a few levelheaded people who give clear, direct, and simple commands.

In summary, then, principles of survival which the Navy *Survival Training Guide* reiterates again and again are as follows:

1. Be stubborn and optimistic, but help yourself.
2. Arrange for basic needs at once.
3. Set up a definite plan with obtainable goals.
4. Don't panic, but keep busy.
5. Recall all you have experienced or heard about survival situations and apply it to the situation in which you find yourself.
6. Hate the situation, not yourself, and fight it.

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AEROSPACE NEUROLOGY

The purpose of this section is to present to the Flight Surgeon sufficient information about certain specific neurological problems with which he may have to deal so that he can make intelligent decisions concerning their management. The subject matter is necessarily restricted, since the Flight Surgeon deals with a population which has been preselected to such an extent that many neurologic problems, particularly those of middle and old age, do not arise. Disorders of consciousness are of critical importance to aviation personnel for obvious reasons.

Epilepsy

Epilepsy may be defined as a paroxysmal neuronal discharge which causes altered consciousness and involuntary behavior, or both. Over half of the patients with epilepsy have no detectable neuropathologic changes in the brain. Frequently, however, it is a symptom of a structural alteration of the central nervous system, so careful attention to personnel at the time of their first seizure is necessary. In the age group with which the Flight Surgeon is likely to be dealing, the most likely diagnosis in anyone who has his first grand mal seizure will be idiopathic epilepsy, although after age 30, the incidence of organic lesions rises. Appropriate diagnostic studies include blood sugar, electrolytes, BUN, calcium, skull X-ray, lumbar puncture, and EEG. The Flight Surgeon should obtain as many of these as facilities permit, and then refer the patient to a center where neurological consultation is available. Anticonvulsant treatment should be started without excessive delay (it is not necessary to wait until the EEG has been obtained). Diphenylhydantoin is the drug of choice for all forms of epilepsy except petit mal.

Epilepsy is incompatible with flying and, in fact, with most military service. Aviation personnel who develop seizures should be grounded permanently. Only if the seizure is clearly the result of an extreme provocation (hypoxia, electrolyte depletion) not likely to recur should one permit such an individual to fly. A question commonly heard is "But does just one seizure make a person an epileptic?" Unfortunately (with a few exceptions, such as mentioned above), the answer is "Yes."

Syncope

Syncope is a more common cause of loss of consciousness and has quite different implications. Syncope is hard to define, either concisely or all-inclusively, but it is not so important to memorize a definition as it is to understand the basic pathophysiology. A careful history is usually much more helpful in providing needed data than is the examination or any lab test. Whereas epilepsy implies an intrinsic brain abnormality, syncope is usually the result of reduced cerebral blood flow. The "simple faint," or vasodepressor syncope, is convenient to consider first. The pathophysiological mechanisms were largely worked out by Engel. A peripheral circulatory collapse, due to loss of vasomotor tone, results in diversion of blood to the skin and muscles. This collapse is mediated through the autonomic nervous system. The result is reduced blood flow to the head. This peripheral diversion of blood is characteristic of response to real or imagined threat, and of mobilization for "fight or flight." Characteristically, vasodepressor syncope occurs in situations where the individual perceives a threat to his safety. External restrictions, usually social, prevent him from actually "fighting or flying," so that there is a failure of the usual compensatory mechanisms to insure adequate cerebral blood flow. Syncope results. It is possible to verify these hypotheses experimentally by blood pressure and pulse recordings. The diagnosis of vasodepressor syncope ordinarily presents no difficulty, since details of the precipitating events and of the spell itself are easily obtained from the history. Syncope in response to specific stimuli, such as injections and the sight of blood, does not make a pilot an unsafe risk. It is not necessary to ground a pilot with this type of syncope. However, a careful evaluation should be made.

Syncopal attacks may occur in the setting of several superimposed stresses on the individual. Such stresses include emotional upset, sleep deprivation, poor nutrition, fatigue, systemic illness, recent alcohol intake, etc. Occasional fainting spells, when one or more of these factors is present, are probably best regarded as "simple faints" and need not result in a pilot's being grounded. Syncope as a result of heart disease (obstructive, arrhythmias) is perhaps the second most likely type to present itself to the Flight Surgeon. In such instances, cardiac output is reduced as a direct result of the obstruction or arrhythmia, so that insufficient blood reaches the brain. Cough syncope is a specific type; it might, perhaps, be better termed Valsalva syncope. Prolonged periods of coughing, laughing, or straining, raise intrathoracic pressure to suprasystolic levels, and thereby block blood flow to the head. Such episodes usually occur in middle-aged or elderly persons with chronic lung disease, but one case has been observed in a 24-year-old Naval Aviator at the Naval Aerospace Medical Institute. It is considered cause for grounding because coughing spells could conceivably occur in flight.

Syncope may result from orthostatic hypotension, which, in turn, may be due either to autonomic insufficiency or hypovolemia. One must actually demonstrate a blood pressure drop, preferably with a tilt table, to establish this diagnosis. Ordinarily, monitoring the pulse rate as well will differentiate between the two conditions, since the pulse slows or stays the same when the autonomic nervous system is affected, but accelerates when hypovolemia is present. True autonomic insufficiency is not likely to be encountered by the Flight Surgeon, but hypovolemia may, since it may result from water and electrolyte depletion or blood loss. Treatment, of course, is directed at correcting the fluid deficit. An isolated syncopal attack clearly attributable to correctable hypovolemia should not interfere with a flying career. Fugue states and dissociative reactions are rarely confused with syncope, since they do not involve the loss of consciousness.

Carotid sinus hypersensitivity may cause syncopal attacks. It is more common in older persons with arteriosclerotic changes in the region of the carotid sinus.

In older indviduals, basilar artery insufficiency is more common and may cause syncope in the form of shorter "drop spells," wherein the patient drops rapidly to the ground as though struck by a club, but with very brief and possibly indeterminate loss of consciousness. This is rare under the age of 45.

"Convulsive syncope" is a term which has been used to describe a phenomenon which has features of epilepsy and of syncope. The attack consists of loss of consciousness, tonic spasm lasting up to 30 or more seconds, perhaps followed by a few clonic jerks, with a rapid return to normal awareness. These spells occur when excessive positive G forces are applied to an individual maintained in a seated or erect position. This positioning prevents compensation for the reduced cerebral blood flow, with the result that an attack of "convulsive syncope" ensues. This has more in common pathophysiologically with syncopal attacks than it does with grand mal epilepsy and the prognostic implications are those of syncope. Attacks of this sort, occurring clearly in response to excessive G forces, should not interfere with a flying career.

Headache

Headache is man's most common bodily complaint. As such, it is important for all physicians to be able to approach the problem of headache rationally. Most headaches can at least be explained, if not relieved, if one is aware of the pain-sensitive structures of the head and neck and the conditions in which they are likely to be affected so as to cause pain. Much of the knowledge in this area is available as a result of the work of the late Harold Wolff.

Muscle contraction (so-called ***tension***) ***headache*** is the most common variety of headache and is one which nearly everyone experiences from time to time. It is generally felt as a dull ache, and is generalized in its distribution. Symptomatic treatment is partially effective, but the patient is done the greatest service by an understanding and concerned physician who takes time to discuss these problems. Frequently, simple verbalization is all the patient may need. Regular interviews, directed at providing simple supportive psychotherapy, are of great value and do not require specialized psychiatric training. The Flight Surgeon is frequently in an ideal position to provide this sort of help, and its potential importance to squadron personnel is obvious.

The second major type of headache is vascular headache, that is to say, ***migraine.*** All physicians who deal with patients will have to deal with migraine headaches. Many physicians themselves are sufferers. The pain of vascular headache comes from the extracranial blood vessels, particularly the external carotid artery. The arterial wall undergoes a change, the nature of which is not known, which makes it dilate excessively with every pulse beat. The resulting pain may be excruciating. Almost all vascular headaches may be regarded as variants of migraine (hypertensive headache is the most significant exception). These headaches possess one distinctive feature—they throb, pulsate, or pound. Most patients are extraordinarily vivid in describing their throbbing pain. Classic migraine is a specific subtype. The headache is unilateral (but not always on the same side) and is preceded by symptoms of cerebral or ophthalmic ischemia lasting 15 to 20 minutes. These symptoms vary widely to include hemi- or quadrant-anopsia (most common), unilateral paresthesia or weakness, aphasia, monocular blindness, and ophthalmoplegia. Characteristically, there is no headache until these symptoms subside; then it strikes (usually contralateral to the symptoms) and may last from hours to days.

Cluster headache is another common type of vascular headache. It is, at its worst, perhaps the most excruciating head pain experienced by man. It is unilateral, usually involves the face, and comes in clusters of individual attacks, each lasting minutes and, less commonly, hours. Clusters may last for days or weeks, headache-free periods of one to three years are common. The sporadic and variable natural history makes it difficult to evaluate treatment. Vasoconstrictors will abolish most of the individual attacks. There is no conclusive evidence that histamine desensitization is of benefit.

The remainder of the vascular headaches may conveniently be grouped under the term *common migraine*. These headaches are frequently bilateral, may or may not be preceded by ischemic symptoms and are highly variable in severity. The pulsating nature of the headache may not always be an outstanding characteristic.

Persons with migraine headache frequently possess certain specific personality traits. They are compulsive people who are striving and perfectionistic. They often set for themselves unrealistic goals and are unable to delegate tasks, preferring to see to every detail personally. They are intolerant of persons less highly motivated than themselves. They frequently find themselves worrying about things over which they have no control. Migraine headaches frequently occur immediately after periods of intense activity, when a letdown is experienced. Weekends and vacations are common times. They may, however, occur during stressful situations.

Management of all types of vascular headaches is the same. The most effective and desirable approach to the patient with migraine is to define his personality traits; if the specific traits mentioned above are discovered, an attempt should be made to develop insight on his part so that he may alter them somewhat. Once this is done, rather superficial directive interview techniques should be employed, telling him, in effect, to "relax and enjoy it." Frequently, one or two interviews of this sort will dramatically reduce the number and severity of the attacks and, occasionally, cures are achieved.

The most useful pharmacologic adjuncts are vasoconstrictive drugs (particularly ergot alkaloids), as might be expected from the pathophysiology.

Methysergide (Sansert) is another useful drug but is perhaps best reserved until other approaches have been exhausted.

The disposition for aviators with migraine is dependent on the type of migraine. Ischemic symptoms, such as hemianopsia, hemianesthesia, and hemiparesis, are incompatible with flight. Student aviators should be deflected from aviation. Designated aviators should be found not physically qualified, unless one is successful in altering the course of the syndrome. Common migraine, on the other hand, need not interfere with an individual's aviation status, unless the attacks are unpredictable in onset and prostrating in severity, or if regular medication is required.

Muscle contraction and vascular headaches are by far the most likely types to be encountered by the Flight Surgeon. It should be remembered that such headaches are usually part of a long-established pattern for dealing with life. They are unlikely to develop in middle age. In other words, a 38-year-old Marine major who develops severe vertex headache of sudden onset is unlikely to be suffering from tension headache.

Head Injury

Head-injured patients may present problems in acute and chronic management. The Flight Surgeon should be able to deal with most of these problems. Trauma may damage the nervous system in many ways. The initial shock effect of the blow itself may result in concussion, contusion, or laceration. Delayed effects such as cyst formation and generalized myelin degeneration may occur. Intracranial hematomas may form and, lastly, epilepsy may arise. The severity of a head injury is best measured by the length of the amnesic period after the injury (posttraumatic amnesia). Retrograde, or pretraumatic, amnesia is not a reliable parameter. Although details of the period of unconsciousness may be lacking, one may always find out the length of posttraumatic amnesia by simply asking details of the patient. Generally speaking, concussion is diagnosed if there is any amnesia before or after the trauma. The presence of a contusion is implied by blood in the spinal fluid. Laceration is ordinarily manifested by focal neurologic deficit. It is, however, not so important to attach a diagnostic label as it is to be able to assess neurologic function accurately.

Diffuse neurological impairment may follow head injury. The predominant sign is impairment of motility, manifested by slowing or awkwardness of movement. Finger tapping, other rapid, rhythmic alternating movements, gait, running, and hopping on one foot are convenient types of performance to evaluate. Pathological reflexes such as the snouting, sucking, and Babinski signs should be tested for. If any motor impairment is present in aviation personnel, they should be found not physically qualified since considerable brain damage is necessary to be detected clinically. Brain-damaged persons not only perform poorly, but have much less reserve, and any sort of stress is not well tolerated. Neurological consultation and followup are ordinarily appropriate after moderate or severe head injury.

Epilepsy may be caused by trauma, but figures of incidence vary widely. Penetrating missile wounds are followed by a 45–50 percent incidence of development of seizures. The best available study on posttraumatic epilepsy following blunt head injury is by W. B. Jennett, an English neurosurgeon. His series of over 1,000 cases, with minimum four-year followup, provides much useful information. His figures indicate that the overall incidence of posttraumatic epilepsy is about ten percent, but that certain categories have a much higher incidence, while others have a much lower incidence. Twenty-eight percent of those patients who had a seizure in the first week following the injury developed seizures at some later time, almost always later than 11 weeks. Twenty-one percent of those patients who suffered a depressed (not linear) skull fracture developed epilepsy; this figure rose to 36 percent if the dura was penetrated. Twenty-eight percent of those patients with intracranial hematoma developed epilepsy. Perhaps more important, of those who had none of these three complications, only one percent developed seizures. More precisely, if the posttraumatic amnesia was less than 24 hours, the incidence was one percent. If the amnesia was more than 24 hours, the incidence was 1.5 percent.

Hence, aviation personnel who, as a result of a head injury, have (a) a seizure in the first week posttraumatic, (b) depressed skull fracture, or (c) an intracranial hematoma should be permanently grounded. It has seemed reasonable to the Division of Psychiatry and Neurology at the Naval Aerospace Medical Institute to allow all others to fly, since the one percent incidence is of the same order of magnitude as the 0.5 percent to 0.75 percent incidence in the general population (see table 6–1).

TABLE 6–1

Incidence of Posttraumatic Epilepsy	*Percent*
Overall incidence	10
Patients with early fit	28
Patients with depressed fracture	21
Patients with hematoma	28
Patients with none of these and with less than 24-hour PTA	1
Patients with none of these and with more than 24-hour PTA	1.5

Vertigo

Vertigo may be defined as illusory rotational movement of the environment and is commonly described by patients as a sense of "spinning," or "whirling around," or "like being on a merry-go-round." It should always be distinguished from aviator's vertigo, which is really spatial disorientation. It is caused by abnormal stimulation of the vestibular apparatus or its central pathways.

Ménière's disease is the most common cause of vertigo. The symptoms are vertigo, tinnitus, and deafness. Characteristically, the vertigo is explosive and frequently disabling in onset and accompanied by tinnitus. The deafness is of a specific type, involving low tones first, but more important, discrimination of speech and perception of pitch are impaired more than would be expected from the pure-tone hearing loss. Abnormal caloric tests are the rule. It is a disease of middle life (third, fourth, and fifth decades, primarily). One must have evidence for both vestibular and cochlear involvement to be sure of the diagnosis, although both may not be evident early in the course of the disease. Pathologically, the characteristic finding is a dilatation of the endolymph-containing portions of labyrinth and cochlea. Ménière's disease is disqualifying for flight.

Many persons experience vertigo without auditory symptoms. Such persons are frequently told they have "labyrinthitis." This implies inflammation of the labyrinth and is not supported by autopsy evidence. A more appropriate term is vestibular neuronitis, since there is evidence that the vestibular nerve or vestibular nuclei are the affected structures, rather than the peripheral labyrinth. Caloric tests are almost always abnormal. One should restrict this diagnosis to patients who have no evidence, historically or audiologically, of auditory dysfunction. This disease is incompatible with safe control of aircraft.

About ten percent of patients with multiple sclerosis develop vertigo as an initial symptom with no evidence of other neurologic involvement at the time of the attack. Multiple sclerosis can only be considered, not diagnosed, at this point.

Vertigo may be a sequel of head injury. Pathological neuronal changes in the vestibular nuclei have been observed following head injury in experimental animals.

Positional vertigo, as the name implies, is vertigo which develops with changes of head position which are characteristic for each afflicted individual. It is ordinarily easily elicited if the physician tests for it. A lag of several seconds follows assumption of the position before vertigo and nystagmus develop.

Attacks of vertigo during flight could, of course, result in disaster. Since most diseases which cause vertigo, unfortunately, are likely to be chronically recurrent, although in some instances with long symptom-free periods, it is the policy at the Naval Aerospace Medical Institute to regard the symptom as disqualifying for flight. It is true that some individuals have single, isolated attacks, but there is, as yet, no way to select out this group.

Summary.

One point stands out: The history is the most important aid to diagnosis of the neurological disorders considered here. Although the ability to perform a skillful neurological examination is useful, the Flight Surgeon should, in most instances, be prepared for appropriate diagnosis and treatment after talking with the patient. As a general rule, commonsense, plus a working knowledge of the natural history of the maladies considered here, will allow the Flight Surgeon to make appropriate dispositions on the logical basis.

APPENDIX

THE MANY FACES OF AIRSICKNESS

BY CAPT ROGER F. REINHARDT, MC, USN, HEAD, DIVISION OF PSYCHIATRY AND NEUROLOGY, NAVAL AEROSPACE MEDICAL INSTITUTE

CASE ONE: The 20-year-old NavCad wears a worried look along with his parachute as he leaves VT–1 Ready Room at Saufley Field, trailing his fast-walking instructor by three paces. It is 1300. Although this fourth presolo flight had been scheduled for 0630 launch, the weather had canceled the early takeoff. When the weather cleared, his instructor had to make an off-wing hop. Over a gulped lunch, the student's knowledge of the day's procedures was discussed and found somewhat wanting. Two previous flights, PS–1 and PS–3, were accompanied by nausea, one by vomiting and a headache which lasted most of the evening. The pair approach their T–34 (lovingly referred to by some as the Maytag Messerschmitt of the Training Command); gas fumes are dancing over from the nearby gas truck. The student grabs his belly, a nearby wingtip, and upchucks freely over the ramp.

CASE TWO: The 23-year-old lieutenant (jg) flight student, only five weeks from his wings of gold, is on his back, but not flat. He's at 21,000 feet in an F–9 trainer out of Chase Field, under the bag, and his instructor has flipped him over into inverted flight, returning control just as approach control comes on the air with approach instructions requiring immediate action. Among the many urgent messages clamoring for the student's attention is a new one: he has to vomit! And pressed to his face is a mask with a hot mike, but with no burp bag attachment.

CASE THREE: Naval Aviator Lieutenant X was an old tailhook man with over 1,000 jet hours, and had just reported to his current assignment from an F–8 squadron. This is his first flight as advanced NAO instructor in the cabin of a T–39. The shades are down, the radar operative, the air smooth, the student attentive, when Lieutenant X stops in midsentence, mumbles, "Excuse me," and heads for where he remembers seeing the brown greasy paper of a burp bag. A minute later, he stumbles forward, replaces the pilot in the left-hand seat, places the wheel in his hands as a look of contentment envelops his face, and completes the flight, never feeling better.

These three cases, ranging the spectrum, illustrate three of the faces of airsickness in naval aviation.

Any discussion of the nature of airsickness should start with the organs of balance and position sense which are closely related to the hearing apparatus (figure 6–3). In the inner ear, organs called the semicircular canals send the message that one is experiencing angular acceleration in the various planes. Similarly, organs called the utricle and saccule send a message when one is accelerating or decelerating along a linear path. Over 100 years ago, it was noted that deaf mutes and others whose inner ears have been destroyed by disease are immune to motion sickness of any variety. A research team from the Naval Aerospace Medical Institute at Pensacola has recently confirmed, off the stormy shores of Nova Scotia, in what was for many a memorable, if unusual, ferryboat ride, that persons without functioning inner ears never become airsick. The Russians were seriously considering destroying surgically the inner ears of their first astronauts to insure that they would not be disabled by motion sickness during the earlier orbital flights.

But other things are implicated besides inner ear sensitivity to acceleration. For instance, the symptoms of motion sickness can be produced by having a person sit in a chair or cockpit, stationary, having the walls of the room twirl rapidly around him. Visual confusion or positional uncertainty, then, may bring the symptoms on. It has long been known that airsickness aloft is usually worse under instrument conditions, and this type can be at least partially alleviated by establishing visual contact with the ground.

Usually, it is the psychological component of airsickness which is most striking. CAPT Philip B. Phillips, formerly Head of the Division of Neuropsychiatry at the (then) Naval School of Aviation Medicine, long ago coined the term, "Emotion Sickness," for the airsickness we see in flight students. Some people are literally not able to ***stomach*** some situations. If we start early in life, we find that there are many infants who show their unhappiness over a changed formula or late feeding, different feeding methods,

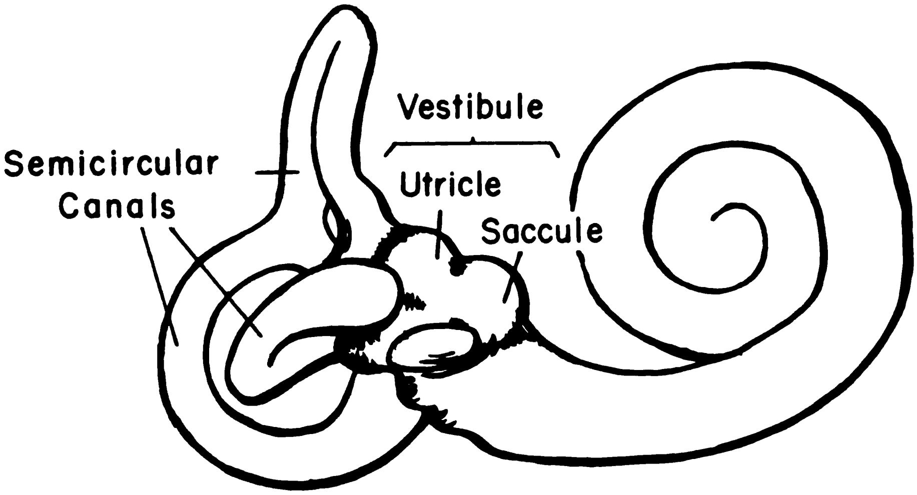

FIGURE 6–3. *Schematic of ear.*

or a different person feeding it, by upchucking the whole thing. There are many six-year-olds who cannot stomach the thought of leaving mama for the first day at school. There are those high school and college students who regularly vomit before every important examination (sometimes they show other evidences of irritable intestines!). There are some who cannot stomach the invisible instructor in the rear cockpit who is, it seems, ever ready with quick and often biting criticism and sarcasm and nearly perfect in the art at which the student feels so stupid. Sometimes the fear of carrying out newly learned procedures, sometimes the positional uncertainty of a spin or other acrobatics; sometimes there is too much G force, and it's new and frightening. Sometimes there's too little G force and it also is new and frightening. In general, we feel that emotional stress is an indispensable condition for airsickness.

The following findings spell out the scope of the airsickness in naval air training:

—Twenty percent of all flight students become airsick to the point of nausea sometime during the first ten flights, so that, for at least a few seconds, they cannot "drive" the aircraft. This is a large figure: one in five.

—More than 97 percent of these, however, become symptom-free and adapted to flying.

—Adaptation occurs, however, only within the first ten flights or the first twenty hours.

—It follows, then, that no flight student should be eliminated for airsickness prior to about his tenth flight, else those might be eliminated who would adapt.

—Recurrent airsickness is a ***dual-instruction disease.*** One should not say "never," but flight students just don't vomit when they are flying solo.

Let us look at the situation first at VT–1, Saufley, that measuring scale in so many ways for fitness for military flying. There are from 10 to 15 drops a year from VT–1 due to airsickness. This represents approximately three percent of all airsick individuals, and approximately 5/10 of one percent of all the total student population which streams through Saufley Field. It is of interest to compare this to World War II Army records which showed that 52 percent of all student pilots who suffered from airsickness washed out before completing their training. When a flight student consults him for airsickness, the Flight Surgeon uses the following approach:

—He listens to the student, acknowledges the student's problem, and tells him that most people get over it. He discusses the difficulties created with some people by having "big brother" always looking over your shoulder and ready always to remind you (although in the kindest tones, of course) of any wrongdoing.

—He is assured that this is a very common problem.

—He is told that considerable tension is natural when one is learning many new procedures rapidly, which must not only be recited on the ground, but converted into flawless muscular action in the air.

—Medication is usually given only when the student has had medication suggested by his instructor. Long experience has proven the antimotion sickness drugs to be of little value in this situation.

Illustrative of the VT–1 management of airsickness is the following exchange of memoranda concerning a student referred to the Flight Surgeon for airsickness by the student's instructor:

First Memorandum (from Flight Surgeon to instructor)—

"The below-named student was interviewed this date in regard to airsickness on his first two hops, and again later on his fifth hop. The latter was flown after a five-day period of bad weather. It is my feeling that this student will develop a tolerance provided he can fly three to four hops weekly. He was not given any medication at this time. We would like to see him again after three or four hops if the motion sickness persists. Thank you. /s/ Lieutenant ______________________ Flight Surgeon."

Second Memorandum—one week later (from instructor to Flight Surgeon)—

"Subj: Second Lieutenant ______________________'s airsickness

1. I have to admire student's ability to continue the hop after having been sick.
2. Student becomes extremely mad at himself for becoming sick.
3. As you see from sick sheet, student has been sick on every hop but one. On most hops he is sick twice and sometimes more. On four occasions, he has not had time to get his bag and has had to clean up the cockpit on return.
4. If he can stand it until PS–11, so can I.

Very respectfully,

/s/ ______________________"

What superb spirit! Unfortunately, this particular student couldn't stand it until PS–11, but the system, as the figures indicate, is a good one.

Airsickness is not really a *major* problem in naval air training past VT–1, and that is as it should be—VT–1 is where the eliminating should be done. About one case every two months is seen among students commencing their jet training at NAAS, Meridian. Airsickness problems in advanced phases of jet training seem to bring into the open about two disabling airsickness cases per year for reasons that are not entirely clear. This far along in the program, two months or less from their wings, the loss is expensive and it hurts.

It is useful to visualize a model for airsickness in which the development of clinical airsickness requires the accumulation of 100 points, from whatever source. As in figure 6–4, if you have only 25 points, you feel only uneasiness. At 75 points, your performance is impaired, while at 100 or more, you are not able to control the aircraft at least for a brief period of time. In the average case we see at the Naval Aerospace Medical Institute, we feel that the spread of points is somewhat as shown in figure 6–5. Twenty of the points probably represent an inherited sensivity of the semicircular canals. There has to be some predisposition factor, for there are some individuals whom no amount of emotional stress can cause to have nausea and vomiting. For this component, which might be called "canal sickness," there is no treatment, and it may even be that there are some individuals, although not in naval aviation, who have 100 points from this source alone. The next 40 points can be visualized as having been accumulated during one's psychological development from infancy to maturity. These people have mislearned and misused vomiting as a solution to problems and conflicts. The final 40 points can be seen as representing current situational difficulties. This may be fear, or doubt of one's proficiency, or depression at the thought of having too much expected of one, or religious confusion about whether it's ethical to fly aircraft

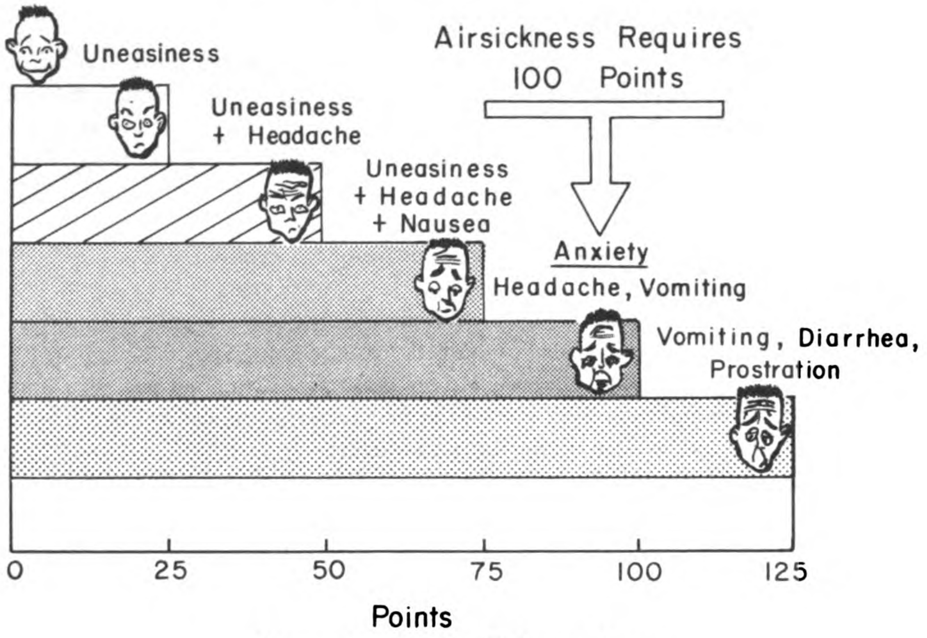

FIGURE 6–4. *A model for airsickness.*

which are in essence lethal weapons platforms, or occupational confusion—maybe he thinks he should have been a priest or a doctor or a fish camp operator.

A look at the figures shows that approximately 20 student naval aviators are lost to the Training Command each year, with the presenting symptom of airsickness. This is much better than in World War II, most of the loss occurs early in the program, and it may be approaching an irreducible minimum.

For the occasional Fleet pilot who develops airsickness, it is probably the return of an old acquaintance (to say an old *friend* would be going too far). For this man, we would give this advice:

—Check your physical condition. Poor fitness, poor diet, overweight, too much boozing, too little sleep—all of these things can trigger a *latent* airsickness response. If this checklist doesn't locate your trouble, consult your Flight Surgeon for a deeper search.

—Check your mental condition. An upcoming deployment combined with a sick or unhappy wife, debts up to your gray hairs, a close-knit family pestering you to come back home to Wire Grass, Alabama—any of these things can tip the scales. If you find something, discuss it with your Flight Surgeon. Sometimes he can help you find alternatives you didn't know existed. If you don't find anything, let him help you look.

—If in a single-engine, two-place aircraft, and the other pilot has the stick, key the mike and say, "I have it." (If the other pilot is senior, perhaps, "Would you like me to take it for a while, sir?" would be better.) If you're special crew in the cabin

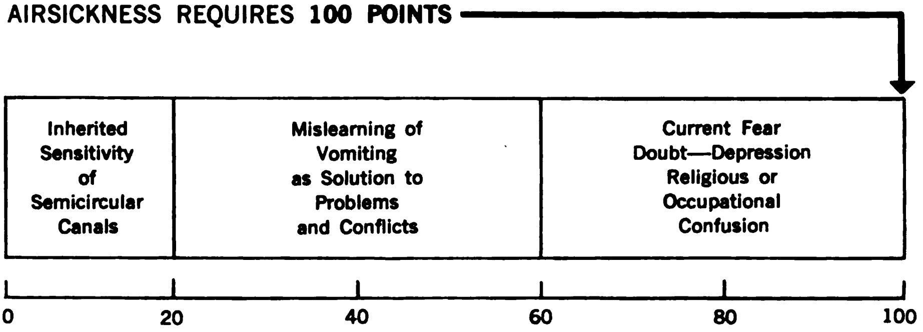
AIRSICKNESS REQUIRES 100 POINTS
Inherited Sensitivity of Semicircular Canals
Mislearning of Vomiting as Solution to Problems and Conflicts
Current Fear Doubt—Depression Religious or Occupational Confusion
0
20
40
60
80
100

FIGURE 6–5. *Spread of points for airsickness.*

of a multiengine aircraft, go forward and hint that it would be nice to get the feel of the controls—the view from there will help anyway. If you can't do this, get where you can see outside and look for visual reference. If you can't even do this, then you're in for a bad few minutes—for you have one of the unhappier faces of airsickness.

CHAPTER 7

Otorhinolaryngology in Aerospace Medicine

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OTOLOGY

- GROSS ANATOMY
  - The External Ear and Auditory Canal
  - The Tympanic Membrane
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  - The Inner Ear
  - Organ of Corti
- EXAMINATION OF THE EAR
- DISEASES OF THE EAR
  - Frostbite
  - Otohematoma
  - Acute Perichondritis
  - Sebaceous Cysts
  - Foreign Bodies
  - Diffuse External Otitis and Otomycosis
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  - Aerotitis Media

HEARING

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  - Malingering
  - Audiometry
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- The Military Aviation Noise Environment
  - Noise in Flight
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- Protection From Noise
  - Environmental Control
  - Protective Devices
- The Navy Hearing Conservation Program
- Hearing References

THE NOSE AND PARANASAL SINUSES

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Examination of the Larynx
Diseases of the Larynx
REFERENCES

OTOLOGY

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GROSS ANATOMY

The External Ear and Auditory Canal

The external ear consists of two parts, the auricle and that part of the auditory apparatus which helps to collect sound waves, and the auditory canal which directs the waves toward the perceptive apparatus. Loss of the auricle does not reduce auditory acuity; the auricle in man is more an adornment, without an important function. The auricle (pinna) is an oblong, flat, molded structure consisting of skin supported by a framework of elastic cartilage. The auricle differs in size and shape in different individuals but both ears are usually symmetrical. Landmarks (figure 7–1) include the helix, antihelix, tragus, antitragus, concha, lobule, incisura intertragica, auricular fossa, and the cavity of the concha (cavum concha) leading to the meatus of the external auditory canal. The cartilage of the auricle is continuous with that of the canal except anteriorly and superiorly where it is completed by a tough fibrous membrane extending between the tragus and the commencement of the helix, the circular, folded outer edge of the pinna.

The *external canal* (external auditory meatus) consists of a cartilaginomembranous and bony portion. It is about 1¼ to 1½ inches in length. (Posterior superior wall is 24mm, anterior inferior wall, 31mm, average.) (See figure 7–2.)

The *cartilaginous* part forms about one-third of the entire length, its direction being slightly upward, backward, and medial. The outer orifice of the canal is elliptical with its greatest diameter directed from above downward. In its lower anterior wall there are several fissures in the supporting cartilage, the incisurae Santorini, which allow for the flexibility of the canal. These fissures are of particular interest because it is through them that

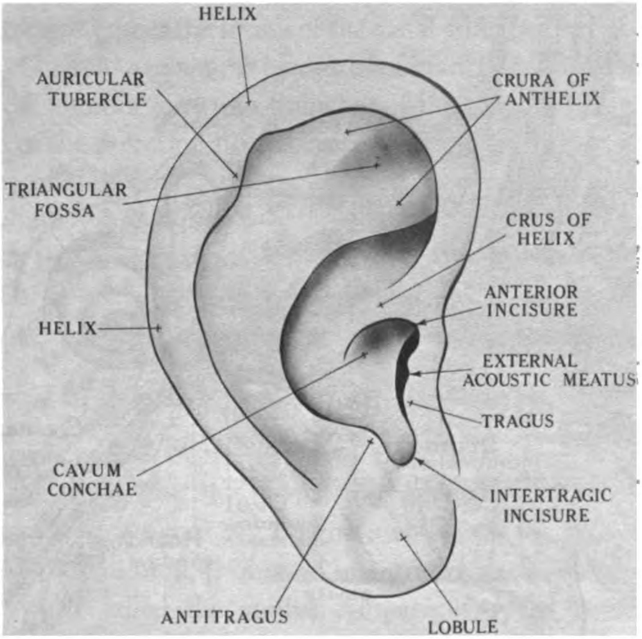


FIGURE 7–1. *Topography of external ear.*

infections of the external canal may extend and descend into the neck below, in front, or behind the pinna (more commonly in very young children).

The *bony canal* (forming the inner two-thirds of the length of the canal) fuses laterally with the cartilaginous portion. Its direction is downward, inward, and forward, forming an angle at the junction with the cartilaginous portion. This angular junction explains the inability to see the tympanic membrane by looking directly through the cartilaginous portion of the external auditory meatus. (See figure 7–2.)

The *epidermal lining* of the external canal is a continuation of the integument of the auricle. Normal skin appendages, hair follicles, sebaceous and ceruminous glands, are found in the outer one-third only (cartilaginous portion). They are absent in the inner bony two-thirds. The skin of the bony canal is tightly adherent to the periosteum, with almost no intervening connective tissue layer. Because of these differences in the epidermal covering of the external canal, furunculosis of the canal occurs only in the outer one-third, a significant factor in the differential diagnosis of infectious processes of the canal and in such things as mastoid infections with a sagging of the bony canal.

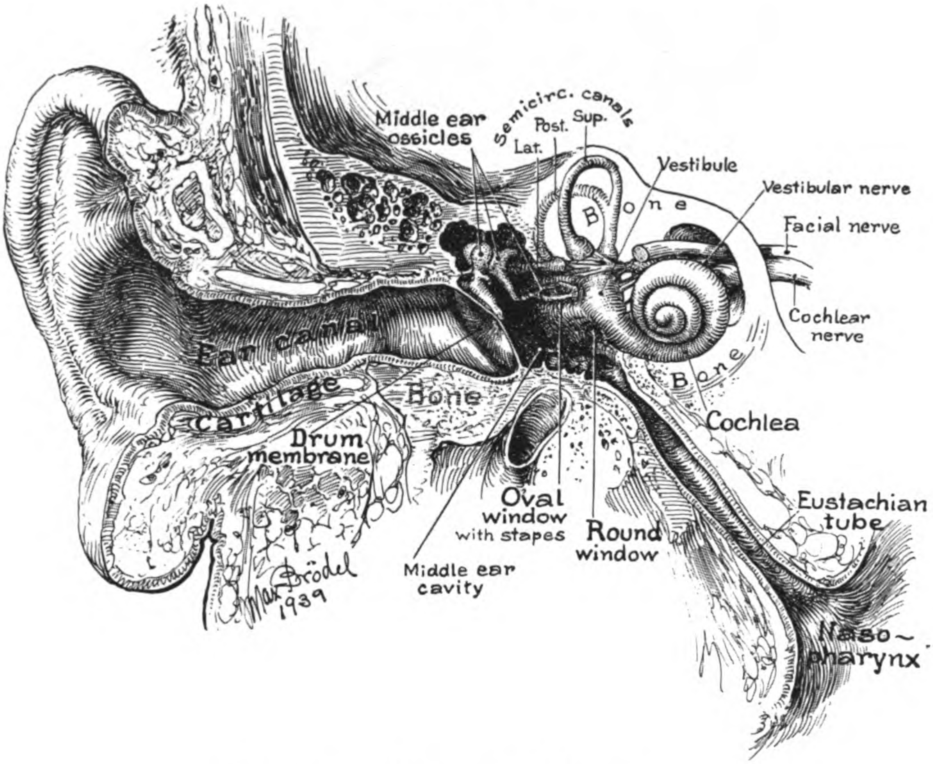


FIGURE 7-2. *Cross section of the entire ear.*

The sensory *nerve supply* of the external auditory canal is derived from the auricular branch of the vagus (Arnold's nerve), and the auriculotemporal branch of the trigeminal (mandibular division). In addition, the auricle derives some of its sensory innervation from branches of the great auricular and the small (or lesser) occipital nerves. These nerves have indirect connections with other nerves so that irritation or disease of the ear may create referred symptoms into remote parts of the body. Also, irritation in other areas such as tongue or teeth may produce pain in the external auditory meatus via the auriculotemporal branch of the trigeminus. Similarly, irritation of the external canal by mechanical manipulation may cause cough because of connection of the auricular branch of the vagus nerve in the brain stem with the motor nuclei of the vagus and allied nerves.

The *arterial blood supply* of the external ear is by twigs from the neighboring superficial temporal and posterior auricular arteries, which are branches of the external carotid artery. The deep auricular, a branch of the internal maxillary artery, supplies the lining of the canal and also part of the tympanic membrane. *Venous drainage* is by means of branches of the posterior auricular vein in the anterior auricular branches of the temporal vein. Because of the position of the posterior auricular veins, there is at times a connection by means of the mastoid emissary vein with the transverse sigmoid sinus.

The abundant *lymphatic plexus* of the auricle and external auditory meatus empties into the preauricular, postauricular, and inferior auricular lymph nodes. These in turn empty into the deep cervical plexus.

The Tympanic Membrane

The tympanic membrane is the lateral membranous portion of the middle ear and, at the same time, the medial membranous portion of the external auditory canal. (See figure 7–2.) The membrane, roughly circular, about 8mm wide and 9mm high, is inserted in a groove in the tympanic bone by a circular ligament called the anulus tympanicus. The eardrum is about one-tenth of a millimeter in thickness and made up of three layers: the outer or lateral layer is a continuation of the epidermal lining of the canal; the inner layer is a continuation of the mucous membrane lining of the middle ear cavity; the middle layer consists of connecting tissue fibers, both circular and radial, which are attached peripherally to the margins of the bony ring and centrally are inserted into the handle and short process of the malleus. This relatively heavy middle layer (pars tensa) is present throughout the membrane except for a small triangular area above the short process of the handle of the malleus, lying in a niche in the superior wall of the canal where the fibrous bands are absent. This part of the tympanic membrane is called Shrapnell's membrane (pars flaccida). Just above the short process of the malleus, internal to Shrapnell's membrane, is Prussak's space or pouch, a frequent site for retraction. The topography of the tympanic membrane (drum head) should be recognizable to all examiners. (See figure 7–3.) The most depressed point of the tympanic membrane is located slightly inferiorly and posteriorly to its center. This area corresponds to the tip or spatula end of the manubrium hammer handle and is termed the umbo. There is approximately a 2mm distance between the umbo and the head of the stapes and the promontory which is the medial wall of the middle ear and represents the basilar turn of the cochlea. If one follows the manubrium anteriorly and superiorly from the umbo, it will be noted to end in a slight projection laterally which is produced by the short process of the malleus. Extending

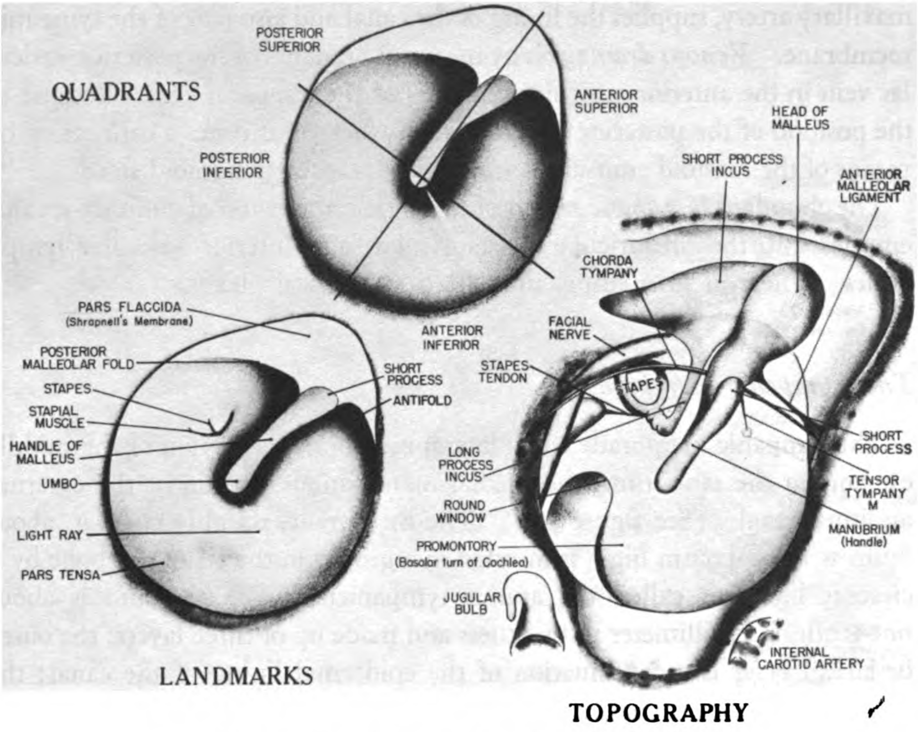


FIGURE 7–3. *Tympanic membrane topography.*

anteriorly and posteriorly from the short process are two ligamentous folds, the anterior and posterior malleolar folds. These folds are of clinical significance because they are definite landmarks which separate the two parts of the drum and when the drum becomes drawn inward, are accentuated. The amount of retraction of a tympanic membrane can be estimated by the prominence of the short process and malleolar folds and by the retraction (foreshortening) of the malleolar handle. Anteriorly and inferiorly from the umbo is the light reflex produced by the concentration of light which is reflected from the concave surface of the tympanic membrane onto its convex surface opposite the region of the Eustachian orifice within the middle ear. Normally the light reflex begins at the umbo as the deepest part of the membrane and never reaches the margin of the tympanic membrane. The condition of the light reflex may be changed, exaggerated, or dulled by disease. The angle of inclination of the tympanic membrane in the adult is such that the posterior-superior portion lies nearest the observer and courses anteriorly and inferiorly to form approximately a 45° angle with the inferior wall of the external auditory meatus. (See figure 7–2.)

The *blood vessels* of the tympanic membrane lie chiefly with their nerve fibers in Shrapnell's membrane, along the posterior margin of the handle of the malleus and just on the margin of the pars tensa. The main supply is derived from the deep auricular branch of the internal maxillary artery, which supplies primarily the cutaneous layer of the eardrum. The mucous membrane surface is provided by the anterior tympanic branch of the internal maxillary and by the stylomastoid branch of the posterior auricular artery. The two sets of arteries communicate by anastomosis through the tympanic membrane. The venous drainage is either into the veins of the tympanic cavity or into those of the external auditory canal.

Although the exact regions of the tympanic membrane supplied by the various nerves are not definitely known, it is certain that they lie in the same regions as the blood vessels. The main nerves are the auricular branch of the vagus (Arnold's nerve), the auriculotemporal branch of the trigeminus, and the tympanic branch of the glossopharyngeal (Jacobson's nerve).

The Middle Ear

The middle ear contains the mechanism for the conduction of sound waves to the internal ear. It consists of a series of pneumatic spaces which include the Eustachian tube, the middle-ear cavity itself, and the cells of the mastoid process. (See figure 7–4.) The cavity of the middle ear is best described as a six-sided box measuring 15mm from above downward and from front to back. Laterally, it is much narrower, measuring about 4mm from side to side near the top, even less than that near the center, and then widening a bit near the bottom.

Lateral Wall.—The lateral wall of the middle ear is partly bony and partly membranous. The membranous portion (the tympanic membrane), discussed above, seals the middle ear cavity from the external auditory canal. The remainder of the lateral wall is the lateral attic wall which is the medial extension of the outer table of the squamous portion of the temporal bone, after it has curved medially to form the roof of the external bony canal. This lateral wall extends from the Eustachian tube to the antrum of the mastoid process.

Superior Wall.—The superior wall (roof or tegmental wall) varies in thickness from 3 or 4mm to tissue-paper thin. It separates the tympanum from the middle cranial fossa. This area is a favorable site for extension of middle ear infections and fractures.

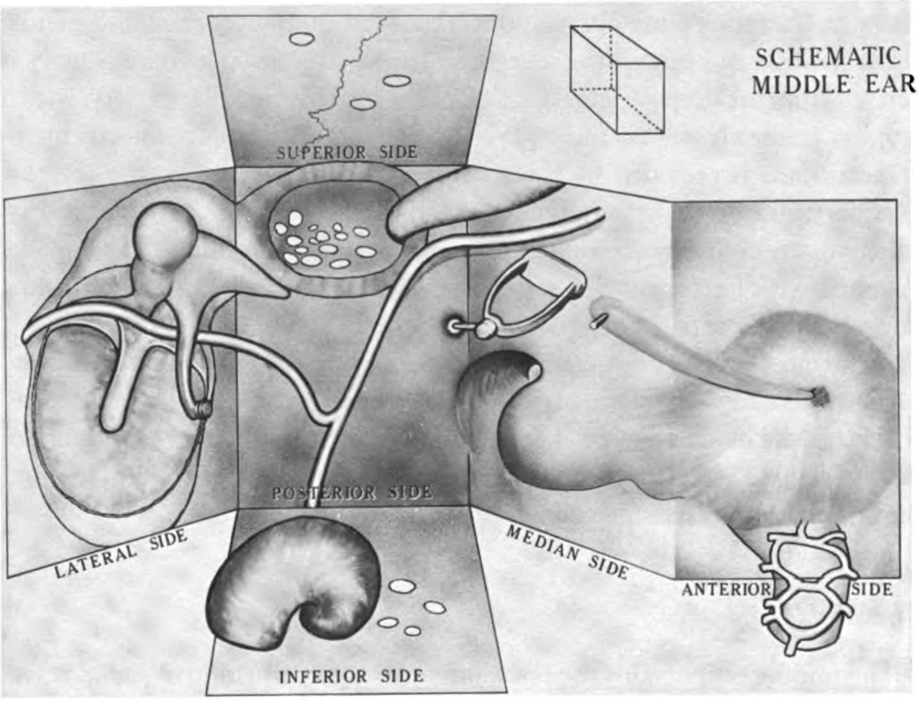


FIGURE 7-4. *Schematic of middle ear* (AFTER LEDERER, 1947).

Inferior Wall.—The floor or inferior wall is also known as the jugular wall. At this point there is a thin bony structure separating the hypotympanum or floor of the typmpanic cavity from the jugular bulb. The bulb may mound into the middle ear cavity; there may be dehiscences in this bony covering in infants. This possibly should be kept in mind whenever a myringotomy is being done, as serious hemorrhage may result if the bulb is nicked.

Anterior Wall.—The anterior or carotid wall of the middle ear contains the tympanic opening of the Eustachian tube, the bony canal of the internal carotid artery, and the semicanal of the tensor tympani muscle. The Eustachian tube connects the tympanic cavity with the nasopharynx. It is approximately 37mm (1½ inches) long and its direction from the tympanum is forward, downward, and inward. In the adult the pharyngeal opening is about 15mm lower than the tympanic end. The tube is one-third bony and two-thirds cartilaginous, the narrowest part of the canal being at the junction of the two portions. The Eustachian tube (osseous portion) opens into the middle ear in the upper half of the anterior wall of the tympanum. The pharyngeal portion of the membranocartilaginous structure forms two-thirds of the entire length and is closed except during the act of swallowing or yawning when its lumen is temporarily widened by the contractions of the levator and tensor veli palatini muscles, mainly, the latter. The osseous portion is always open.

Posterior Wall.—The posterior or mastoid wall is a direct continuation of the posterior wall of the external canal; it is distinguished because it contains the facial canal embodying the facial nerve (descending portion) and arteries. The facial canal lies approximately 3mm posterior to the external auditory meatus wall and exits from the skull through the stylomastoid foramen. In the posterior wall are branches of the middle meningeal artery from the internal maxillary artery by way of the mastoid foramen. Supplying the mastoid cells is the auricular branch of the occipital artery. Also from this wall is the tensor tympani muscle with its motor nerve from the facial nerve.

Medial or Labyrinthine Wall.—The medial wall is also the lateral wall of the inner ear. It contains several important structures and niches. (See figure 7–3.) These structures include the facial canal, the oval window containing the footplate of the stapes, the promontory, and the round window. Anterior to the horizontal portion of the facial canal is the processes cochleariformis, a pulleylike structure about which the tendon of the tensor tympani muscle curves before inserting into the neck of the malleus. The oval window, closed by the anular ligament and the footplate of the stapes, communicates with the perilymph space of the vestibule. Below it in a deep depression lies the round window. It is closed by a membrane, the secondary tympanic membrane which separates the middle ear cavity from the scala tympani of the first turn of the cochlea. The *promontory* is a plate of bone which bulges into the middle ear cavity representing the first (basilar) turn of the cochlea. The facial nerve lies in a canal above the stapes, below the bulge of the horizontal semicircular canal. The entire cavity is covered with mucous membrane. The mucosa is more elevated anteriorly at the mouth of the Eustachian tube and is ciliated.

Tympanic Cavity Proper and Auditory Ossicles.—The tympanic cavity contains the ossicular chain of the sound-conduction system. Within the tympanic cavity there are three small, movable bones: the malleus, incus, and stapes. These auditory ossicles extend like a chain across the tympanic cavity, the better to connect, functionally, the tympanic membrane with the vestibular (oval) window. The outermost (lateral) ossicle is the malleus, firmly attached to the tympanic membrane; the innermost, the stapes, is fixed into the vestibular window and is in direct contact with the fluid perilymph of the inner ear; intermediate between these lies the incus. The three bones are bound together by articulations, and also are attached by ligamentous connections with the walls of the containing cavity. This compound osseous system acts like a bent lever to convert vibrations of the tympanic membrane into intensified thrusts of the stapes against the perilymph.

The muscles within the cavity include the tensor tympani (innervated by the trigeminal nerve through the tensor tympani branch from the otic ganglion) and the stapedius muscle (the smallest of all skeletal muscles), which arises in the interior of the hollow of the pyramidal eminence on the posterior tympanic wall. Its tendon escapes through the opening at the apex and then turns inferiorly to insert in the posterior surface of the neck of the stapes. It is innervated by the stapedial branch of the facial nerve. The two muscles are antagonists which act as dampers to prevent excessive movement of the ossicular chain during excessive stimulation. The body of the tensor tympani muscle lies in a semicanal above the Eustachian tube. Its fibers arise from the bony walls of this canal.

Mastoid.—As was noted previously, the posterior wall of the middle ear cavity is a continuation of the posterior wall of the external canal. Superiorly, the posterior-lateral wall of the middle ear contains the opening into the mastoid antrum, the aditus ad antrum, which leads into the mastoid antrum. On the medial wall of the mastoid antrum, just posterior to the aditus, the horizontal semicircular canal forms an eminence. The mastoid antrum is really a large air cell which is formed before birth by upward and posterior expansion of the tympanic cavity. The mastoid air cells form from this site by further pneumatization of the mastoid bone. The mastoid process, containing a tip cell, is formed by the union of processes of the squamous and petrous portions of the temporal bone. Pneumatization of the mastoid varies in different individuals, but all the cells communicate with the antrum and all are lined with mucous membrane similar to that of the middle ear cavity.

The Facial Nerve.—The facial nerve is a structure with which one is constantly concerned. Its anatomy should be considered in fairly close detail, not only because it is in close proximity to the internal and middle ear and mastoid process, but also because it serves as an indication of the progress of infection within and without the temporal bone. (See figure 7–5.) The nerve follows a tortuous path, receiving and sending many branches in its course from the brain to its final distribution to the muscles of expression of the face. Grossly, the nerve blends with the pars intermedia (or glosso-

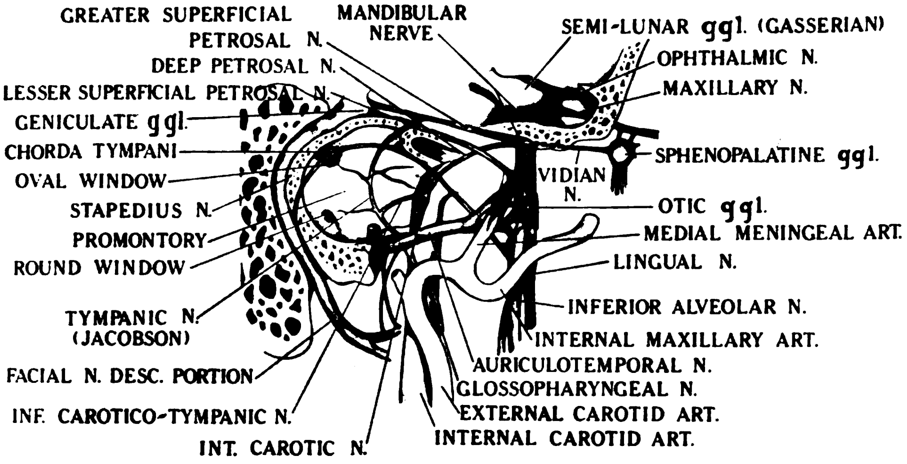


FIGURE 7–5. *Facial nerve relationships* (AFTER LEDERER, 1947).

palatine nerve) which contains both parasympathetic and sensory fibers, while it is still in the internal auditory meatus. (See figure 7–6.) The nerve enters the internal auditory meatus coursing laterally and forward to the facial knee (first portion). The geniculate ganglion is located at this point. From here the nerve bends backward, traversing the middle ear, above the oval window as far as the posterior wall of the middle ear cavity (intratympanic portion). At this point the nerve bends sharply downward to cruise down through the mastoid tip and emerge from the stylomastoid foramen (descending portion). Branches of the facial nerve given off within the facial canal are:

1. The great superficial petrosal nerve which leaves the facial nerve at the genu and passes into the sphenopalatine ganglion via the Vidian canal.

2. Anastomotic branch with the tympanic plexus, a fine thread, from the facial knee to the lesser superficial petrosal nerves.

3. In its downward course the facial nerve gives off, *first,* a stapedius nerve which arises in the upper portion of the facial canal and passes through the sievelike wall to supply the stapedial muscle within the eminentia pyramidalis and *second,* the chorda tympani nerve, found beneath the stapedius nerve, and courses back in an acute angle, opened above, going through the chorda tympani canaliculi to the tympanic cavity. Here it lies in a posterior malleolar and anterior malleolar fold between the long process of the incus and the handle of the malleus. It passes through the petrotympanic fissure in order to arrive at a position medial to the angular spine, middle meningeal artery, and the auriculotemporal and inferior alveolar nerves.

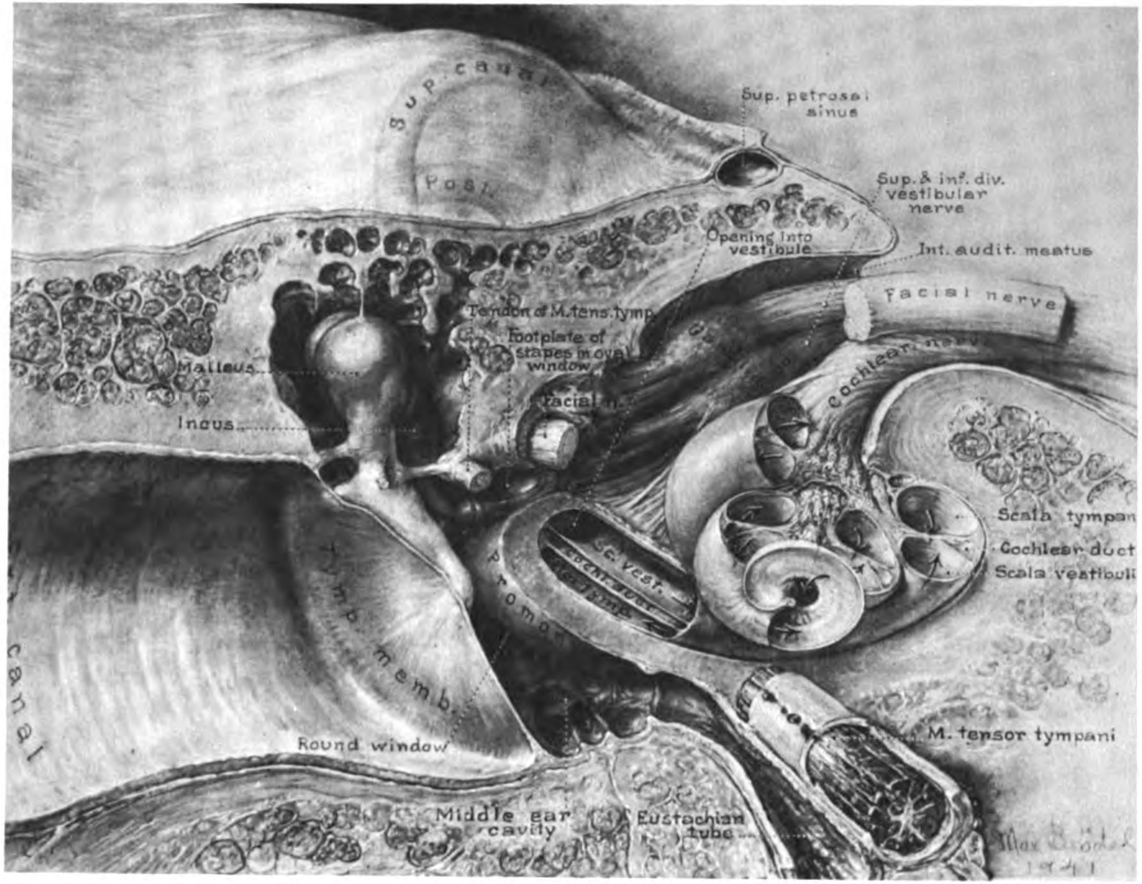
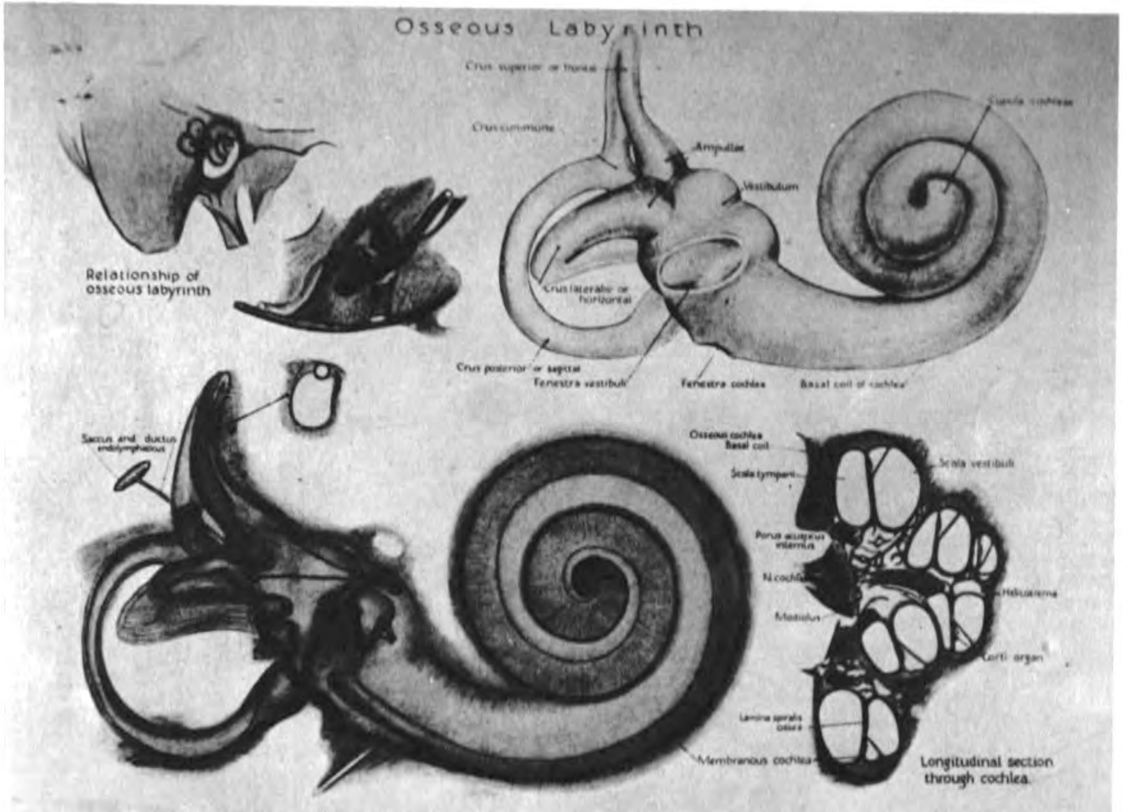


FIGURE 7-6. *Relationships of the middle and inner ear.*

The Inner Ear

The internal ear consists of two portions supplied by two distinct sensory nerves; namely, the ***vestibular labyrinth*** with its vestibular portion of the eighth nerve, and the *cochlea* with its cochlear portion of the eighth cranial nerve. The entire structure is located in the petrous portion of the temporal bone which completely surrounds the inner ear except at certain communications with outer surfaces. The osseous internal ear or bony labyrinth consists of the bony semicircular canals, vestibule, and cochlea (figure 7–7). Laterally to the internal ear is the cavity of the middle ear; medially, the internal auditory meatus. Through the latter, the inner ear connects with the posterior cranial fossa. The bony labyrinth capsule of the petrous pyramid consists of very dense bone, 2 to 3mm thick. The bony inner ear has five exits: (1) oval window (closed by the stapes), (2) round window (closed by a membrane), (3) cochlear aqueduct, (4) vestibular aqueduct, and (5) internal auditory meatus. Within the bony labyrinth is a membranous counterpart, the membranous labyrinth. This is surrounded by supporting fluid and is anchored to the walls of the bony labyrinth by numerous small trabeculae. The supporting fluid is called perilymph. The membranous labyrinth itself is filled with endolymph. The inner ear is separated from the middle ear by the oval and round windows. Physiologically, the labyrinth contains two entities, the acoustic apparatus, consisting of the cochlea and perhaps the saccule, and the vestibular apparatus which consists of the three semicircular canals and the utricle. (See figures 7–8 and 7–9.)

The Cochlea.—The bony cochlea is a short cone lying medial and slightly anterior to the middle ear cavity. The apex of the cone points anteriorly and laterally. It consists of 2½ turns about a central bony core, the modiolus. It is about 9mm in diameter and 5mm high. The membranous cochlea is divided into three sections by the basilar membrane and Reissner's membrane. The three divisions are (1) the scala vestibuli which communicates via the vestibule with the oval window, (2) the scala media which contains the organ of Corti, and (3) the scala tympani which is separated from the middle ear by the round window membrane. The scala vestibuli and scala tympani, containing perilymph, communicate by a small opening at the apex of the cone, the helicotrema. The scala media contains endolymph and is not in connection with the perilymph of the scala vestibuli or scala tympani. The organ of Corti, bathed in endolymph, is the sensory end organ for the reception and conversion of sound impulses and is connected



The osseous internal ear consists of the bony semicircular canals, vestibule and cochlea. It lies within the petrous portion of the temporal bone, adjoining laterally the labyrinthine portion of the tympanic cavity, and medianward the floor of the internal auditory meatus (indicated by the relationship of the osseous labyrinth in a transparent right temporal bone viewed laterally and from above, together with the eighth nerve, internal carotid artery, jugular bulb and eustachian tube). The bony structure almost completely surrounds the delicate membranous internal ear.

FIGURE 7–7. *The bony labyrinth.*

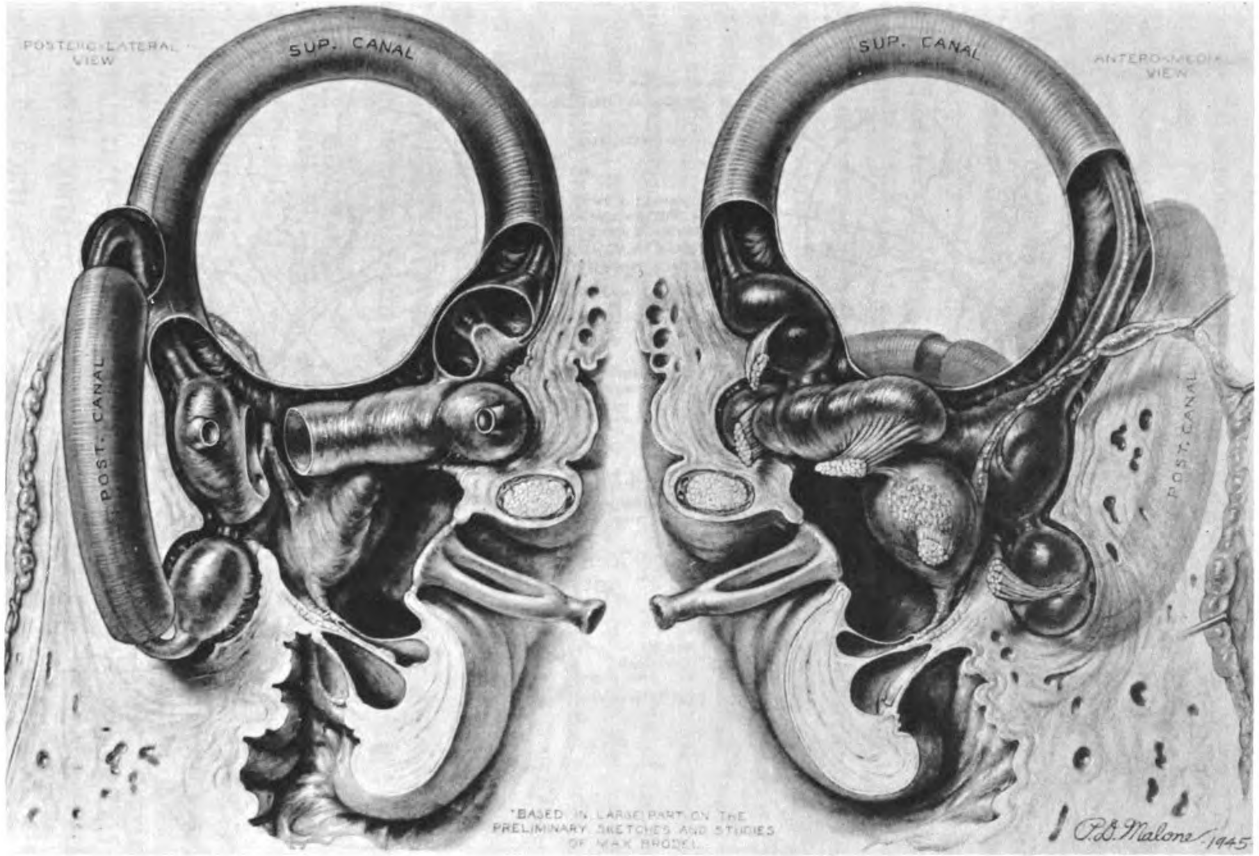


FIGURE 7–8. *Semicircular canals.*

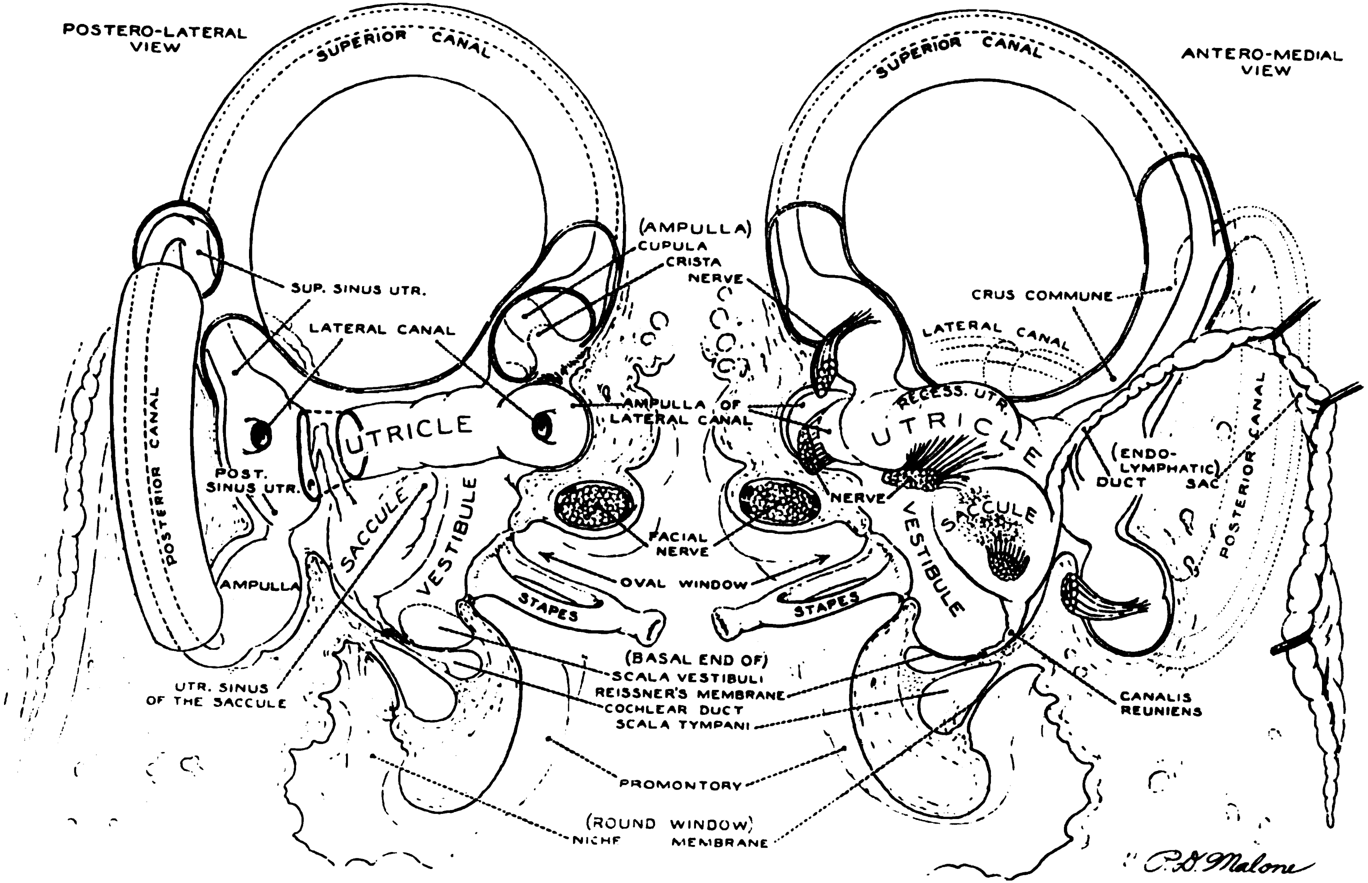


FIGURE 7-9. *Semicircular canals (schematic).*

with the cerebral centers by the auditory VIIIth nerve. (See figure 7–10 for semidiagrammatic representation of the cochlear duct.)

Vestibule.—The vestibule is a small cavity separated from the middle ear by the oval window. It contains two irregular saclike organs, the utricle and the saccule. (See figures 7–8 and 7–9.) The utricle connects with the membranous semicircular canals and forms part of the vestibular apparatus. It also communicates through a narrow channel with the saccule, which in turn joins indirectly with the scala media or ductus cochlearis. The structure and function of the nonauditory labyrinth are discussed in detail in chapter 8.

Semicircular Canals.—The semicircular canals are curved tubes, three in number, nearly semicircular, each of which is connected in two places with the wall of the vestibule. (See figures 7–8 and 7–9.) They are not all of equal length but are almost uniform in diameter (1.5mm). They lie in three planes almost perpendicular to each other, any two forming a right angle. The horizontal canal is the shortest of the three and is directed laterally and backward. Laterally and forward it adjoins the recess epitympanicus and there produces the prominence of the horizontal canal in the floor of the antrum above the horizontal portion of the facial nerve. The superior semicircular canal stands at right angles to the axis of the petrous portion of the temporal bone, making an angle of 45° with the sagittal axis of the skull. It is close to the surface of the bone, giving rise to the prominence in the middle cranial fossa called the eminentia arcuata. The posterior semicircular canal is the longest of the three. It lies almost parallel to the posterior surface of the petrous bone.

The membranous semicircular canals correspond in shape to the bony canals but are of much smaller diameter. They join the utricle through the

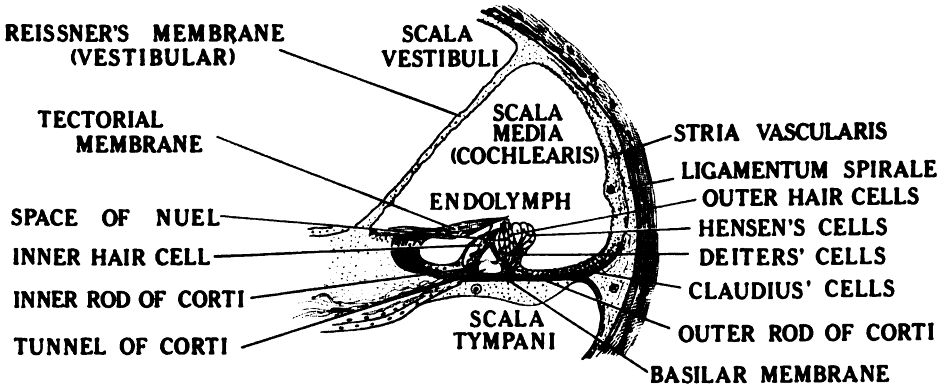


FIGURE 7–10. *Transverse section of the cochlear duct.* (FROM Diseases of the Ear, Nose, and Throat (2D ED.) BY W. W. MORRISON, COPYRIGHT 1955, APPLETON-CENTURY-CROFTS.)

five openings in the walls of the vestibule. There are but five openings as the posterior and superior canals join and enter the utricle through a common opening, crus-cummunions.

The Utricle and Saccule.—The semicircular canals comprise what is called the kinetic portion of the labyrinth. The utricle forms the static portion. They lie, as has been noted, in the cavity of the vestibule between the cochlea and the semicircular canals. They are small membranous sacs, the utricle being the larger of the two. The utricle serves the five openings of the membranous semicircular canals. It also has on one side an oval, thickened place of whitish color, the macula acoustica utriculus, in which terminate the fibers of the utricular nerve. The saccule is a flattened spherical area (2mm in diameter) situated in front of and below the utricle. It communicates directly with the scala media of the cochlea. On the medial surface there is a thickened area in which terminate the fibers of the saccular nerve. The saccule is connected with the utricle only indirectly through the endolymphatic duct. This duct ends in a blind cecal dilation under the dura of the posterior surface of the petrous bone, the endolymphatic sac.

The Cochlear Nerve.—The nerves of the cochlea and the labyrinth run together until they reach the medulla but from there they diverge widely. The cochlear nerve has reflex connections in the brain stem but it is mainly connected with the cortex of the temporal lobe of the same and opposite side. Recent experimental work indicates that the cochlea on one side sends an equal number of fibers to both temporal lobes—a symmetrical bilateral cortical representation. After entering the medulla, the cochlear fibers end about the cochlear nuclei which are in close relationship to the inferior cerebellar peduncles. From these nuclei some of the fibers run dorsally across the floor of the fourth ventricle to the lateral fillet of the opposite side. Others run ventrally as the trapezoid body to the lateral fillet and ascend to the inferior corpora quadrigemina and medial geniculate body of the opposite side. Some fibers go to the lateral fillet of the same side; some to the nuclei of the nerves to the extraocular muscles. These latter are responsible for the associated movement of the eyes in response to sound. From the inferior quadrigeminal body and the medial geniculate body, fibers pass to the auditory center in the cortex of the temporal lobe. Numerous decussations connect the various centers on the two sides. Because of the symmetrical bilateral representation of each cochlea in the brain it is rare to find deafness associated with any lesion in the brain situated above the level of the cochlear nuclei. The entire temporal lobe on one side can be removed without causing an appreciable hearing loss. From this and a mass of other experimental data it follows that a lesion causing hearing loss is situated in or peripheral to the cochlear nuclei.

Organ of Corti

The organ of Corti is that portion within the cochlea where the cochlear nerve and its twigs reach their termination. It is contained in what is commonly called the cochlear duct or the scala media. The organ consists simply of several rows of nerve hair cells which are held together by longer and firmer supporting cells which rest directly on the basement membrane. (See figure 7–10.) The tops of the hair cells project just above the level of the tops of the supporting cells (Deiters). Lateral to these are other types of supporting cells, including the fat-containing cells of Hensen. Over the point of attachment of the basement membrane to the bony lamina (the modiolus of the cochlea), two of the supporting cells are modified to form rod-shaped structures called the rods of Corti. These rods are wide apart at their attachment to the basement membrane below; above they are in contact. By this arrangement a tunnel is formed which extends from the base to the apex of the cochlear duct. The hair cells are arranged in three rows external to Corti's rods and one row internal to the rods. Their nerves pass from the base of these cells into canals in the bony lamina labyrinth to the modiolus. There they join the spiral ganglion, then pass into the internal auditory meatus to form the eighth nerve along with the vestibular branches. Just above the hair cells of the organ of Corti is the tectorial membrane. Its histology is still in doubt as is its extent and true function. Some authors believe that it is a gelatinous membrane into which the hair cells of the organ of Corti project, as in the gelatinous cupola overlying the cristae in the ampullae of the semicircular canals. At the base of the cochlea the basement membrane of the organ of Corti is very narrow. It becomes progressively wider toward the apex until at the top coil the individual fibers are about three times as long as they are at the base.

EXAMINATION OF THE EAR

Inspection and palpation of the external ear, the canal, and surrounding structures is important. Palpation of the areas about the ear may reveal swelling of soft tissues over the mastoid process or enlarged and tender cervical lymph nodes. Pressure on the tragus or pulling the pinna upward may cause pain, indicating the presence of an external otitis. To visualize the external canal and the tympanic membrane, it is necessary to have a good light, the rays of which can be projected into the canal parallel to the line of vision.

Occasionally, it is possible to visualize the canal and the tympanic membrane by pulling the pinna upward and the tragus slightly forward. More commonly, it is necessary to introduce an ear speculum of appropriate size, depending upon the size and the shape of the canal. Usually it is not possible to see the entire tympanic membrane without tilting the speculum slightly from side to side. The speculum normally is not inserted past the cartilaginous portion of the canal, or, in other words, it is used just to the inner edge of the hair-bearing area, but it is not normally extended down into the bony canal where insertion would cause pain. An adequate examination of the external ear should include identification of all possible visible landmarks on the tympanic membrane, including the short process, the handle (umbo), cone of light, Shrapnell's membrane, and pars tensa. Only by repeated examinations of normal ears will a doctor learn the variations of normal (see figure 7-3).

DISEASES OF THE EAR

Frostbite

Frostbite is the most frequently encountered thermal effect to the ear, and the susceptibility to this condition varies highly with the individual. Frostbite may occur as an incident in high-altitude flying, and constant vigilance is necessary to prevent its occurrence in aviators engaged in such work.

The sequence of pathologic changes in the tissues affected by cold is not greatly different from that produced by heat, and consists of engorgement of the blood vessels as thawing of the part takes place, followed by stasis of blood in the capillaries, the exudation of serum, and vesiculation. The degree of change in the tissues will be in direct proportion to the length of exposure to cold and the degree of temperature encountered. The victim of frostbite may be wholly unaware of its onset and suffer prolonged exposure because of lack of the warning of pain. In severe cases, necrosis of tissue and loss of substance from sloughing will occur.

The first step in treatment consists in thawing the part and restoring circulation.

The aftermath of a severely frostbitten ear may be not unlike a severely burned ear. The prevention of infection and the preservation of nourishment to the part are the chief objectives to be obtained in treatment. The application of bland ointments is of considerable value. These may contain antibiotic or chemotherapeutic agents.

Otohematoma

The diagnosis of hematoma is easy. Soon after trauma there is rapid effusion of blood between the cartilage and the perichondrium. A round, soft, bluish-red swelling forms quickly on the anterior surface of the auricle, obliterating the contour of the helix and antihelix. If seen early, the contents of the hematoma may be aspirated. If late, it is often necessary to incise and express the clot. A snug dressing after evacuation of the clot will help prevent recurrence. The dressing should be maintained for 36 hours. In any event, some attempt should be made to remove blood or fluid beneath the auricular perichondrium to prevent "cauliflower ear."

Acute Perichondritis

Acute perichondritis may be either a serous or suppurative exudation beneath the perichondrium of the ear. In addition to trauma, poor nutrition and certain infectious diseases have caused the serous type. The suppurative type, frequently with abscess formation, usually has a definite history of infection or trauma. Conservative treatment consists chiefly of supportive measures. Dry or moist heat helps hasten absorption. It is sometimes helpful to aspirate the fluid from the collections under the perichondrium. A snug dressing over a mold of dental compound will help maintain the shape of the ear.

Sebaceous Cysts

Because of the necessity for wearing close fitting helmets and radio earphones by airmen, sebaceous cysts about the auricle should be mentioned. As elsewhere on the body, the treatment of choice is complete extirpation of the sac, after any infection is brought under control.

Foreign Bodies

Foreign bodies may be animate or inanimate, organic or inorganic. Animate foreign bodies include a great variety of insects. Methods of removal vary from holding a light near the ear to spraying ether vapor into the canal. A simple method is to fill the canal with oil, after which the canal is syringed with warm water. In removing other foreign bodies it is important to know whether or not they are hygroscopic. Those which will not absorb water may be washed out. If instrumentation is necessary, the greatest care should be exercised to prevent injury to the thin and delicate skin of the bony portion of the canal. Even impacted cerumen can usually be removed by means of a forceful stream of water. The use of sterile water is indicated, for many a doctor has washed cerumen from an external canal only to find a perforation in the tympanic membrane.

In removing inanimate, inorganic foreign bodies from the canal, especially those situated at or beyond the auditory canal isthmus, it is safer for the physician to get behind the foreign body and rake it out. A copper wire probe with the distal millimeter bent 90° from the shaft can be used as a rake. Thumb and bayonet forceps often slip off a smooth foreign body and chase it down against the tympanic membrane, which may be perforated during additional attempts to grasp the object.

Diffuse External Otitis and Otomycosis

Diffuse external otitis and otomycosis are infections of the external auditory canal, are extremely common, and the morbidity is high, especially in temperate and tropical areas. During the hot summer months, the incidence of this affliction may be higher than the common cold among all cases coming to the Flight Surgeon.

Certain individuals are predisposed to repetitive infections, and such patients almost always prove later to be suffering from faulty cerumen formation. Therefore, soft ear wax that is not excessive to the point of obstruction of the canal, should not be removed. Individuals who have dry, waxfree, pruritic auditory canals should be given bland ointments, such as Borofax or one-percent yellow mercuric oxide to be applied topically to the canals several times a week. Insertion of contaminated fingers or any other object into the canals should be discouraged.

The opening of the swimming season heralds the advent of the auditory canal inflammations, as the introduction of water into the canal lowers the vitality of the skin, macerates it, and makes it more vulnerable to infection. It is not certain whether new bacteria are introduced at this time or whether the bacteria normally present are suddenly allowed to invade. This matter is not important. At any rate, it is now known that these infections are predominantly bacterial and not mycotic. Otomycosis can occur concomitant with bacterial otitis externa, but the bacterial flora generally predominates. The few exceptions to this are easily recognized; moreover the treatment of the differing etiologic conditions basically is the same.

The Pseudomonas aeruginosa (b. pyocyanea) is the most common bacterium recovered in these infections. The fungal strains of Aspergilli and Monilia are most often encountered in otomycosis. Mixed bacterial and fungal contaminations are most commonly found, if the clinical cases are subjected to culture.

The symptoms are pain and discharge, preceded by itching. Loss of hearing occurs if the canal becomes occluded by edema. The pain is often out of all proportion of the physical findings.

Treatment consists primarily of meticulous cleansing, either by dry wiping or by irrigation with three-percent hypertonic saline, in which instance the ear must be carefully dried. If severe edema is present, various medicaments are used on a wick placed in the canal. This helps to carry the medication to the drum which may be completely obscured by the swelling. It matters little which medication is used provided it is not too strong. Repeated cleansing, daily, in some cases, is of paramount importance.

Successful medication in the treatment of external otitis is first directed at the controlling of pain. Inflammatory reaction caused by the infection is best treated by continuous warm, moist compresses about the auricle and in the external auditory canal. From a practical standpoint this is best and most easily carried out by the use of Burow's solution (N.F.). The combination of a cortisone solution and antibiotic, such as Cortisporin, Neo-Delta-Cortef, and other proprietary preparations is of marked advantage in the treatment of external otitis. The cortisone used locally and topically in the ear has a decided effect in the reduction of inflammatory swelling in the ear and thereby in the reduction of pain. The antibiotics commonly included in these preparations are Polymyxin B (which is considered to be a specific for the Pseudomonas infection), Neomycin, and Bacitracin, which are also active on the various organisms which commonly infect the external auditory meatus.

Furuncles of the External Auditory Canal

Furuncles of the external auditory canal usually occur in the cartilaginous portion of the canal. The bony portion of the canal does not have hair follicles, sebaceous or sweat glands—the routes by which infection most commonly enters the skin. Pain is the outstanding symptom. As the skin is closely attached to the underlying cartilage, there is little subcutaneous tissue present. A slight amount of swelling causes tenseness of the area with production of pain out of all proportion to the size of the lesion. Because of this, patients usually seek medical aid early. If the lesion is not obvious, it may be located by touching different parts of the canal wall with a cotton-tipped applicator.

In the diagnosis of furunculosis it must be remembered that any disease which attacks skin elsewhere may involve the external auditory canal. Perhaps the cardinal point in furunculosis is the negligible reduction in hearing. In mastoiditis and otitis media there is marked hearing loss. In furunculosis, tenderness is elicited by superficial pressure on the tragus, or by lifting the auricle, while in mastoiditis the pain is deep-seated and elicited by pressure over the mastoid bone. Roentgenograms are useful in determining bone pathology or cloudiness of the mastoid.

In treatment of furunculosis, first consideration is given to control of the pain. Warm, moist compresses over the auricle, alternating with dry heat, give the best results. After localization, incision of the abscess will hasten resolution. Loose packing of the canal with alcohol-saturated strips of gauze is helpful. In severe cases chemotherapy should be employed. The roentgen ray is often quite valuable in treating furunculosis in its incipiency. With resolution, Cresatin may be used in cleansing, followed by the application of bland ointments.

Injuries of the Tympanic Membrane

Injuries of the tympanic membrane may follow direct trauma such as careless instrumentation or indirect violence such as occurs from a slap on the ear or entering the water incorrectly when diving. It is the primary finding in aerotitis where the causative factor is a difference in air pressure on the two sides of the tympanic membrane. The evidence of injury may vary from the slightest hyperemia to a ragged perforation of the tympanic membrane. In cases of head injury with fracture into the middle ear, the possible complications and consequences are extremely serious. In most cases, however, the treatment consists of watchful waiting. It is not even advisable to attempt to clean the external canal, the possibility of introducing infection into the middle ear being greater than the danger of leaving it alone. Hemorrhages or exudates into the membrane are absorbed spontaneously. The great majority of lacerations will heal by scar tissue formation in a few weeks. Those that do become infected should be treated identically like an acute otitis media with perforation of the tympanic membrane.

Myringitis Bullosa

Myringitis bullosa is an inflammatory process confined to the outer layers of the drum and usually caused by infections of the canal or middle ear. It is characterized by the formation of vesicles is the drum membrane. It is commonly associated with influenza. If the middle ear is not involved, the blebs subside spontaneously. Mild sedation and the instillation of Auralgan into the external canal suffice to control the discomfort. The newer cortisone preparations also are a useful adjunct in the treatment of this disease. Rarely do the chemotherapeutic agents have any effect upon the course of myringitis bullosa.

Otitis Media

In a chapter of this size and scope, such a comprehensive subject as otitis media can be discussed only briefly. In 85 percent or more of such cases the condition is secondary to disease of the upper respiratory system. Perhaps congestion of the mucous membrane of the nasopharynx has caused obstruction of the orifices of the Eustachian tubes. Perhaps overzealous blowing of the nose has resulted in forcing infected material from the nasopharynx into the Eustachian tube itself. The sequence of events is roughly as follows: Obstruction of the Eustachian tube is followed by absorption of the air in the middle ear. The symptoms are stuffiness in the ear, loss of hearing (conductive type), and pain. If not treated at this stage, transudation of fluid into the middle ear follows. If infection follows, the middle ear cavity will fill with pus. If untreated, the tympanic membrane commonly ruptures and pus drains into the external canal. Once drainage is established, resolution may proceed rapidly. The chemotherapeutic agents and broad-spectrum antibiotics have proved wonderfully efficacious in treating acute diseases of the middle ear. The serious complications of mastoiditis, sinus thrombosis, and brain abscess, so common before the advent of these drugs, are now rarely seen.

All cases of acute otitis media with constitutional signs of inflammation should be given adequate doses of an antibiotic or chemotherapeutic agent, and the patient should be kept on the drug until complete resolution occurs. However, in spite of the above, suppurative otitis media must still be considered a form of abscess, and surgical drainage of abscesses is a rule to be enforced even today. Therefore, the minor operation called myringotomy should not be abandoned, especially when one considers the aspects of future hearing. The incidence of deafness has not decreased since the advent of antibiotics. This may be due to our reliance on the antibiotics to resolve these infections completely, which they often do not. A "smoldering" otitis media may hang on for weeks, with the only symptoms being stuffiness in the ear and deafness. The physician may be surprised to find that such an ear can be filled with fluid.

In addition, a tympanic membrane should not be allowed to rupture spontaneously because of accumulating pus in the middle ear. These spontaneous perforations can occur in a region that will highly predispose the middle ear to later cholesteatomatous formation, especially if the perforation occurs in Shrapnell's membrane.

Conservative treatment of acute suppurative otitis media, i.e., with systemic antibiotics, antibiotic eardrops, and intranasal vasoconstrictors (such as Tyzine 0.1 percent) is usually indicated when the physician first sees the patient; if relief and resolution have not been achieved in a reasonable time, paracentesis of the middle ear may well be indicated. Consultation with an ENT specialist should be requested, if available. The use of the Gottschalk needle aspirator will frequently aid the examining doctor in the obtaining of a sterile culture in order to treat more adequately the specific infection with type-specific antibiotic.

Aerotitis Media

Aerotitis media is an acute or chronic traumatic inflammation caused by an atmospheric pressure greater than the pressure in the middle ear. It is characterized by pain, deafness, tinnitus, and occasionally vertigo. It is probably the most common otitic disorder among flying personnel today. In aircraft, and particularly in military aircraft, the body must compensate for rapid changes in atmospheric pressure. At 18,000 feet the atmospheric pressure is

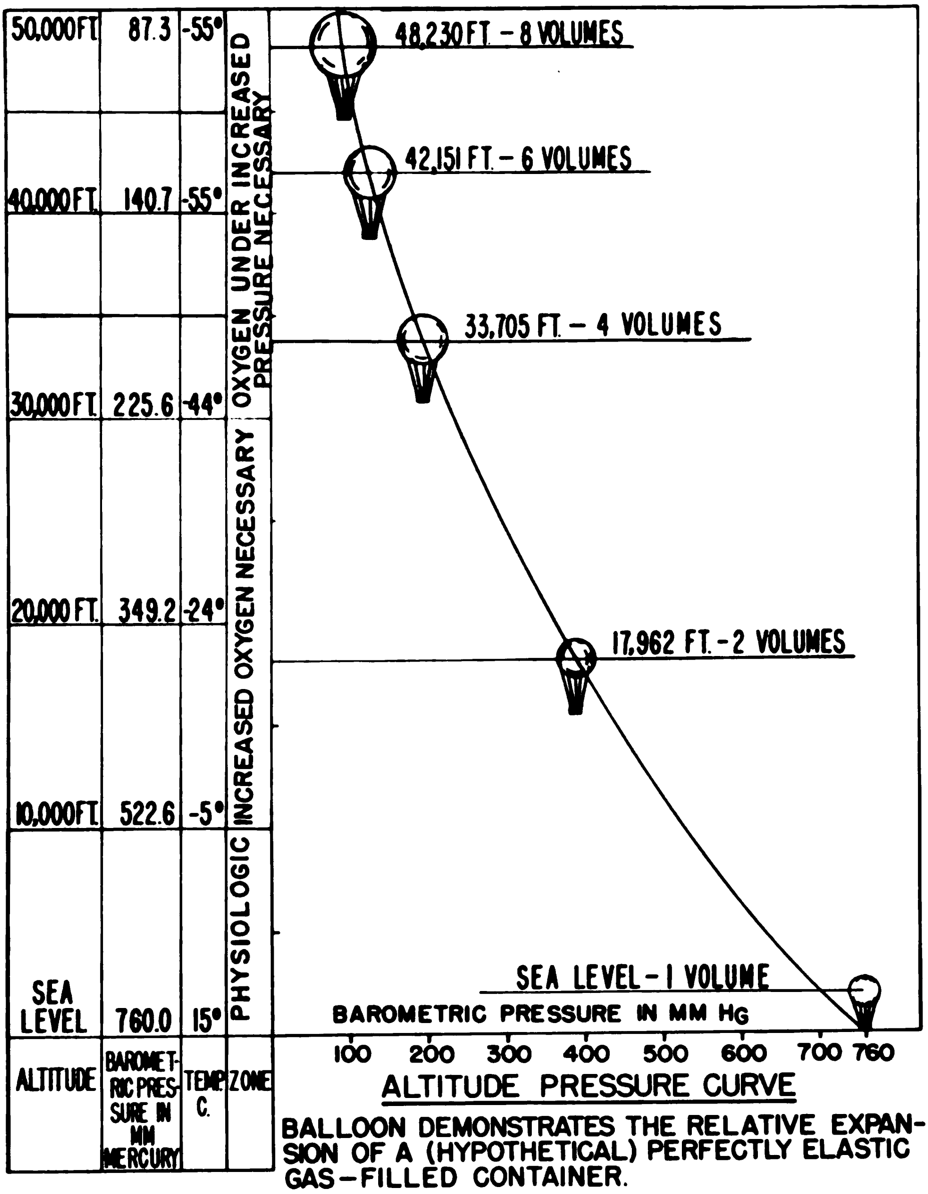


FIGURE 7-11. *Altitude—pressure curve.*

roughly half of that at sea level (760mm Hg). At 33,000 feet it is roughly one-fourth, or only 196mm Hg (figure 7–11).

Present-day aircraft are capable of ascending and descending many thousands of feet per minute. It is essential that there be ready interchange of air between the middle ear and the external air in order to maintain equal pressure on the inside and outside of the tympanic membrane.

Under normal conditions, atmospheric pressure is maintained inside the middle ear cavity through the Eustachian tube. The pharyngeal end of the tube is slitlike in shape and acts as a one-way flutter valve. The lumen is closed except during the acts of yawning, swallowing, chewing, etc. At such times the actions of the levator and tensor palatini and the salpingopharyngeus muscles exert tension on the lower lip of the Eustachian tube, causing it to open.

During ascent, the air in the middle ear expands as the external pressure decreases. A pressure change of only 3 to 5mm Hg (approximately 150 feet at sea level), will produce a slight sensation of fullness in the middle ears, and examination will show the tympanic membranes to be bulging slightly. With an increase in altitude of 500 feet from sea level (15mm Hg), there will be a "click" in the middle ear as the Eustachian tube is forced open by excess pressure in the tympanic cavity, middle ear pressure equalizes, and the tympanic membrane snaps into its normal position. It has been found that the Eustachian tubes continue to open at approximately 425-foot increases in altitude, even up to 35,000 feet, at which point the corresponding pressure change is only 3.5mm Hg. This is probably a function of the increasing ease with which the less dense air at the higher altitude passes through the Eustachian tubes as compared with the denser air at lower altitudes.

When the atmospheric pressure is increased in descent from altitude, a totally different effect is produced. The collapsed pharyngeal end of the Eustachian tube then acts as a flutter valve preventing entry of air (figure 7–12). The aviator must remember to swallow, yawn, or Valsalvalize while descending. When he swallows, the lips of the tubal opening are pulled apart and air rushes into the middle ear, equalizing pressures within and without immediately. This is the usual mechanism by which pilots keep pressure on the two sides of the eardrum equalized during descent to lower altitudes. If, however, the pressure differential exceeds 80mm Hg, the muscles can no longer open the tube. In such cases it is necessary to reduce the external pressure by climbing back to a higher altitude, if possible.

When the aviator is unable to equalize the pressure in his ears by conscious

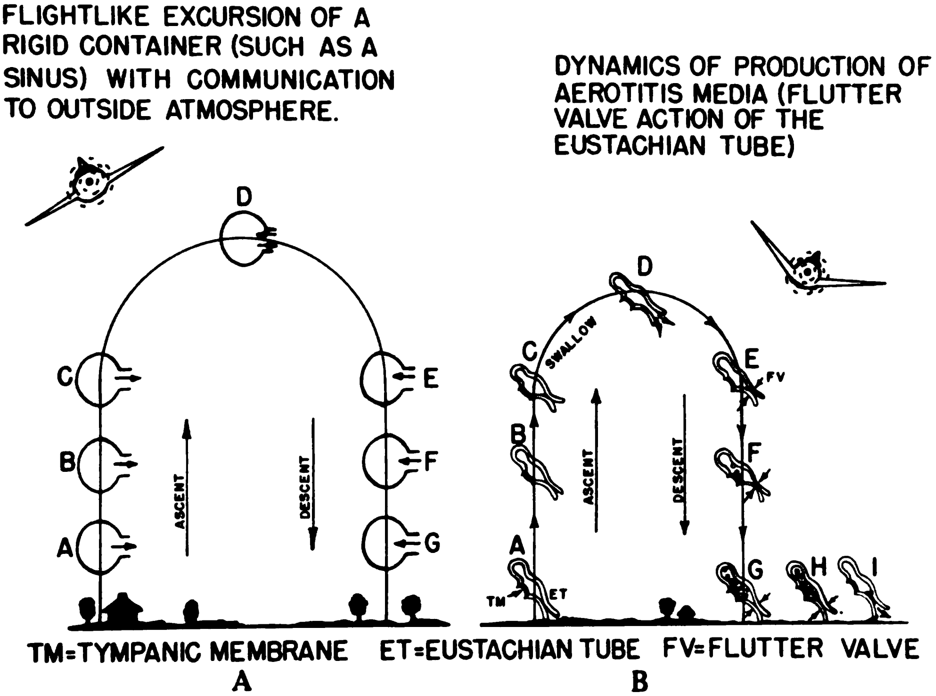

FLIGHTLIKE EXCURSION OF A RIGID CONTAINER (SUCH AS A SINUS) WITH COMMUNICATION TO OUTSIDE ATMOSPHERE.
DYNAMICS OF PRODUCTION OF AEROTITIS MEDIA (FLUTTER VALVE ACTION OF THE EUSTACHIAN TUBE)
D
C
B
A
E
F
G
ASCENT
DESCENT
D
C
SWALLOW
B
A
TM
ET
E
FV
F
G
H
I
ASCENT
DESCENT
TM=TYMPANIC MEMBRANE
ET=EUSTACHIAN TUBE
FV=FLUTTER VALVE
A
B

FIGURE 7-12. *Mechanics of aerotitis media.*

effort during descent, there is a rapid onset of deafness, tinnitus, and pain in the ear. Vertigo may be present. When the differential pressure reaches 60 to 80mm Hg, the pain is severe. At a differential pressure of 100 to 500mm Hg, the membrane ruptures. When this occurs, he suffers excruciating pain associated with a loud explosive noise in the ear, vertigo and nausea become marked, and may be followed by shock and general collapse. Obviously, such a condition is intolerable for aircraft pilots. Recovery is rapid but dull pain persists for a day or so and hearing loss for a longer period.

The findings on examination vary with the amount of pressure differential to which the ear has been subjected. In the less severe cases, there may only be signs of a retracted membrane. These include increased brilliance of the light reflex and backward displacement of the handle of the malleus. Increased prominence will be found of the handle of the malleus and especially of the short process and the anterior and posterior folds. Shrapnell's membrane will be markedly retracted.

In more severe cases there may be hyperemia of the tympanic membrane with small hemorrhages into the membrane itself. The inflammation is most marked along the handle of the malleus and about the periphery. Depending upon the severity of the trauma and upon the length of time the condition has been present, varying amounts of serous or bloody fluid may be present in the middle ear cavity. If the tympanic membrane has ruptured, there will be bloody fluid in the external canal.

Differential diagnosis may be a problem when the patient first reports for examination hours after the onset. The differential diagnosis is tabulated in table 7–1.

TABLE 7–1

Differential Diagnosis of Aerotitis Media, Otitis Media, and External Otitis [1]

	Aerotitis media	Otitis media	External otitis
1	Due to barometric pressure changes	Inflammatory	Inflammatory
2	Retraction of drum	Bulging of drum	View of drum may be obstructed
3	Drum landmark accentuated	Drum landmark obliterated	
4	Rupture of vessels	Diffuse erythema	
5	No thickening of drum	Thickening of drum	May be thickening of drum if visible
6	Usually no fever	Fever usually present	May be fever
7	White blood cell count normal	White blood cell count elevated	White blood cell count elevated
8	Serosanguineous fluid in middle ear	Serous or seropurulent fluid in middle ear	No fluid in middle ear
9	Hearing normal or slightly reduced	Deafness profound	Hearing normal if canal not obstructed
10	No pain on pressure over tragus and movement of auricle	No pain on pressure over tragus and movement of auricle	Pain on pressure over tragus and movement of auricle
11	No swelling of canal	Slight if any swelling of canal	Swelling of canal

[1] From Armstrong, 1961.

Perhaps the most important item in ***treatment*** is prophylactic. Careful examination to insure functional patency of the Eustachian canals before selection for flight training is essential. Men with chronic inflammatory diseases of the nose or paranasal sinuses should be screened out. In the field, men with upper respiratory infections must be grounded until the Flight Surgeon is satisfied that the mucous membranes of the nose and throat have returned to normal. The vast majority of cases of aerotitis media occur among pilots who insist on flying in spite of the presence of an acute upper respiratory infection.

Pilots and passengers must be taught and reminded to equalize pressure on descent and the maneuvers for so doing. Active treatment is confined to the nasopharynx and treatment of the underlying cause. The nose and nasopharynx are sprayed with one of the vasoconstrictors after which attempts should be made to inflate the middle ear.

Equalization of the pressure may be accomplished by the Toynbee, Valsalva, or Frenzel maneuvers, or when necessary, by Politzerization or by means of the Eustachian tube catheter. A drawback of the Politzer maneuver is the fact that it inflates more successfully the ear that is the least blocked, the air trapped in the nasopharynx entering the path of least resistance. *Catheterization of the Eustachian tubes should be performed only by those experienced with the procedure because any rough manipulation of the pharyngeal end of the tube only adds insult to the injury.* It has been found that aspiration of the accumulated fluid in the middle ear by means of a fine needle, under local anaesthesia, considerably reduces the morbidity of aerotitis and shortens the period of convalescence. This should be done by a specialist in ENT if available.

The affected flyer should be kept on the ground until all signs of his upper respiratory infection have disappeared or at least until he can successfully perform the Valsalva maneuver.

Incidence of aerotitis media in Adler's series of thousands of low pressure chamber subjects was 3 to 6 percent, with severe cases about 0.3 percent.

Oxygen Otitis Media.—There is another form of acute otitis media, resembling aerotitis media but different in etiology, of which the Flight Surgeon should be aware. It might be termed oxygen otitis media or perhaps anoxic otitis media. When the aviator has been flying at altitude for some time breathing 100 percent oxygen, especially while using miniature regulators (which supply only 100 percent oxygen), the air in the middle ear is replaced in part by 100 percent oxygen. While descending, the aviator, in equalizing middle ear pressure, replaces any remaining air with more pure oxygen. On completion of the flight, the pressure in the middle ear is equalized with the atmospheric pressure, but the middle ear is filled with 100 percent oxygen.

If the aviator then goes to sleep so that normal yawning and swallowing movements do not occur (thus replacing the 100 percent oxygen with ambient air), the oxygen will be absorbed in the venous blood in rather short order, leaving a moderate to severe vacuum in the middle ear. This is precisely the physical situation in aerotitis media, and as of that moment the aviator has aerotitis media indistinguishable from ordinary aerotitis media except by history.

Oxygen otitis media can be prevented simply by equalizing middle ear pressure during descent with air rather than oxygen. An even better and simpler method is to swallow, yawn, and Valsalvalize several times after descent, so that the oxygen in the middle ear is replaced by air. The difficulties in this method arise from forgetting to do it and from inability to know when sufficient replacement has occurred. Treatment of oxygen otitis media is identical to that of aerotitis media.

Chronic retraction of the membrane tympani is not uncommon. It is most frequently caused by inflamed lymphoid tissue in or about the pharyngeal orifice of the Eustachian tube. Chronic paranasal sinusitis, allergic or otherwise, may be present. Less commonly it may be due to atresia of the canal or closure of the opening by scar tissue adhesions. A carefully done adenoidectomy in childhood is the best deterrent to chronic Eustachian tube obstruction in adults. The patients complain of stuffiness in the ears and are found to have a conductive type of hearing loss. These people almost always have difficulty in keeping the Eustachian tubes open when flying. Repetitive inflations of the middle ear by direct Eustachian tube catheterization have benefited some of these patients, but the problem is always a serious one in flyers, and careful attention to any or all of the underlying factors by the Flight Surgeon is paramount.

HEARING

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Theory and Nomenclature of Sound, Noise, and Hearing

Sound is a physical phenomenon consisting of a wave of pressure variations in various media transmitted in all directions from a source. The wave form of a pure tone sound is sinusoidal. The *amplitude* of the sine wave determines the *intensity* of the sound measured in decibels (db). The *frequency* of the wave determines *pitch* and is expressed in cycles per second, or Hertz (abbreviated "Hz"). A Hertz is a unit of frequency equaling one cycle per second.

Sinusoidal sound is rarely seen in the natural environment. Most sounds are compound waves with a fundamental frequency and "overtones" which bear an arithmetical relationship to the fundamental frequency. The fundamental frequency determines the pitch of the sound, but the overtones give it its *"timbre,"* which allows us to distinguish one voice from another, for example. The three qualities of loudness, pitch, and timbre are those of interest in hearing.

Sound intensity is measured in terms of decibels. The intensity of the faintest sound which can be heard is known as its *threshold,* which varies with individual auditory acuity. The decibel is the minimal identifiable auditory increase at threshold level. *Total intensity* can be measured directly in decibels by a sound pressure level meter. It gives a single decibel reading as a measure of the intensity of a sound, but such a single reading does not adequately describe the hearing hazard. *Frequency band analysis* is the usual method today of measuring sound since it gives the intensity (or sound pressure level, abbreviated SPL) by frequency band or octave band. The ear is much more susceptible to permanent damage by higher frequency sound; therefore, this information is much more useful and important.

Noise has been defined as "any unwanted sound." Since any sound capable of producing harmful effects on the individual is unwanted, noise is the sort of sound with which the Flight Surgeon must deal.

Hearing involves the transmission of sound to the inner ear, the change of the sound wave to a neural impulse, transmission of that impulse to the brain, and perception of the impulse by the brain, i.e., the input of acoustic information to the brain. Every individual has a hearing threshold for every frequency audible to him. This varies considerably among individuals, and in the same individual changes with age. Several other factors must be taken into account, as will be shown.

The auricle in most lower animals is movable and is of assistance in gathering sound waves coming from various directions. In man, who has lost this power of mobility, this organ can be regarded only as the expanded portion of a funnel which is formed by the external auditory canal and terminates in the tympanic membrane. The purpose of this funnel is to direct sound waves toward the tympanic membrane. Although of minor importance because one can hear without the external ear, the role played by the external canal and the auricle is by no means negligible. This is indicated by the assistance derived in certain forms of deafness from speaking tubes or simply by cupping of the hand behind the ear.

The tympanic membrane at the end of the external auditory canal is admirably adapted for the transmission of waves. The area of the vibrating surface varies with the membrane's conic shape and with the degree of firmness of attachment at the anulus. By reason of its obliqueness and slight concavity, the waves are reflected from its peripheral to central portion, where the insertion of the long process of the malleus marks the beginning of the ossicular chain. The latter is to be regarded as a system of levers to convert the vibrations imparted by sound waves to the tympanic membrane into stimuli of sufficient force to produce an appropriate movement in the fluid of the internal ear. Owing to the leverage arrangement of the ossicular chain, the sound waves are transmitted to the cochlea decreased in amplitude but increased in intensity. The tensor tympani and the stapedius muscles, attached to the neck of the malleus and the stapes, respectively, are antagonists, which contract in response to strong stimulation. By decreasing the amplitude of movement of the ossicular chain, they produce a damping effect on the movements of the mechanism of the ear. This reaction has been termed the acoustic reflex. Disease of the nasopharynx which interferes with the patency of the Eustachian tube also interferes with the free movement of the tympanic membrane in the ossicular chain, causing a decrease in auditory acuity. It has been demonstrated experimentally that an intact drum is not absolutely essential for normal hearing. Any interference with the ossicular chain, however, is very likely to result in some hearing loss.

In the human cochlea, localization of perception of high tones in the basal cochlear coil near the oval window has been thoroughly established. The nerve endings of the organ of Corti which lie upon the basilar membrane and insert into the tectorial membrane, are acted upon by the fluid wave carried by the endolymph of the scala vestibuli. The dimensions of these membraneous canals are minute. Some 25,000 to 29,000 ganglion cells, each with numerous hair cell connections in the organ of Corti, are crowded into the spiral cochlear ganglion.

The function of hearing is divided into two separate or general classifications: (1) the conductive apparatus, which consists of the external ear, the tympanic membrane, and the ossicles, and (2) the perceptive apparatus, consisting of the cochlea, the auditory nerve, its nuclei, and the complex cerebral connections of the auditory pathway. Since this highly complex mechanism is concerned with the conduction and perception of sound, it follows that any condition causing interference with the conductive mechanism would result in a conduction deafness. Similarly, a lesion of the perceptive mechanism would result in a perceptive (now referred to as sensorineural) deafness. Lesions in both the conductive and perceptive systems result in a mixed type of deafness. In conductive deafness the hearing loss is more marked in the lower tones. In the classical perceptive type of deafness, on the other hand, high tones are lost first.

THE EFFECTS OF NOISE ON HEARING

Conductive Hearing Loss Due to Noise Exposure

The human ear has a marvelous dynamic range. No known electronic equipment is sensitive enough to respond to intensity levels of 0.0002 dynes/cm^2 (the absolute threshold for sound) and still operate with little or no distortion at intensity levels a ***trillion*** times higher (130 db).

Obviously such a sensitive mechanism requires protective mechanisms to prevent overloading. One mechanism is the ***acoustic reflex.*** A contraction begins in the stapedius muscle about 15 milliseconds after sound stimulation and a bit later in the tensor tympani, although the latency varies markedly with sound intensity. To a prolonged intense sound, the bilateral reflex contraction attains an initial maximum and then gradually lessens, falling to a prestimulation tension in about half an hour. This action causes up to 40 db loss (especially in the lower frequencies) in the energy transmitted to the inner ear, but acts too slowly to protect against explosive noise.

If impact or explosive noise occurs without warning, as is most often the case in war, the protective effects of this reflex will not be available, and damage to the hearing mechanism can result. To give some idea of the time factors involved, the noise profile of an M-1 rifle, when measured one meter away from the muzzle and perpendicular to the barrel, shows that onset of the first positive pressure wave occurs in less than ten microseconds and its pressure level is over 160 db. This is immediately followed by a negative pressure wave of almost equal intensity. Then a period of irregular noise from the gases and the operation of the mechanism follows. The whole event takes slightly over one millisecond.

Such impact noise can cause direct physical damage to the conductive mechanism, including actual rupture of the tympanum. This may heal without sequellae, but concurrent damage to other parts of the hearing apparatus may cause some degree of permanent deafness from the single exposure.

Sensorineural Hearing Loss from Noise Exposure

Just as the protective mechanism of the ear acts to protect it over the short term (up to 30 minutes) with the acoustic reflex, so other adaptive mechanisms are brought into play for longer term exposures to noise. These mechanisms act at the neural rather than conductive level, and the short- and long-term effects of these mechanisms are different from those of the reflex.

Noise-induced hearing loss of the sensorineural type occurs as a temporary threshold shift (or TTS) as measured audiometrically. This is considered, but not proved to be, due to fatigue of cochlear cells. Noise-induced temporary threshold shift (NITTS) can become permanent (NIPTS). This is believed to be due to cochlear damage.

Exposure to intensities in excess of 130–140 db may actually cause sensation in the ear itself, such as discomfort, tickle, and finally pain, as noted in figure 7–13.

The basic problem with which the Flight Surgeon is concerned, however, is noise-induced threshold shift, either temporary or permanent, in flight and ground personnel.

There is a remorseless train of logic which makes such threshold shift a major problem to the Navy and to industry in general.

1. The most disabling hearing loss is that which causes a loss of speech discrimination ability.
2. Speech discrimination is a function of high-tone hearing ability, in the sense that it depends upon hearing 2,000–4,000 Hz.
3. In sensorineural deafness, high tones are lost first and are those more severely affected.
4. Noise-induced deafness is essentially sensorineural type.
5. The frequency which is adversely affected earliest is 4,000 Hz, which is not essential for easy understanding.
6. Therefore, permanent high-frequency hearing loss may be far advanced before the individual becomes aware of it.
7. Indeed, as the hearing loss progresses, noise will bother him less and less and, until he becomes aware of loss of speech discrimination, he will be more, rather than less, content with his noise exposure.

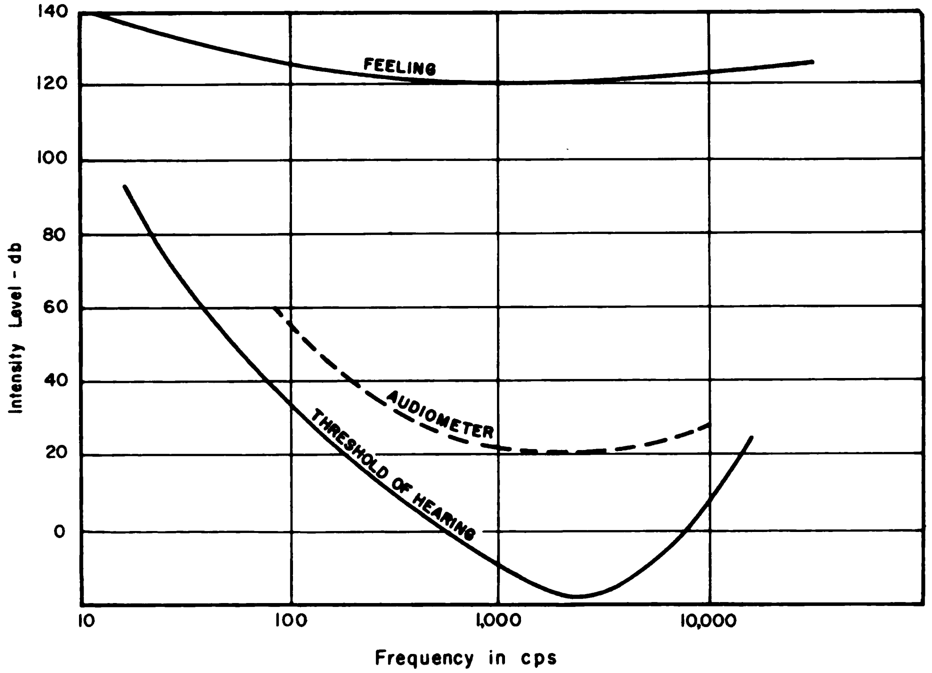

FIGURE 7–13. *Audibility curve in man, showing thresholds of feeling and hearing.* (TOWE & RUCH, 1965—AFTER LICKLIDER, CHAPTER 25 IN Handbook of Experimental Psychology, S. S. STEVENS (ED.). NEW YORK: JOHN WILEY & SONS, 1951.)

Figure 7–14 shows the temporary threshold shifts which occur with exposure to continuous steady noise of noted intensity as a function of log time. Obviously, the auditory threshold shifts upward with time, and the magnitude of the shift is related both to exposure time and exposure sound pressure level (SPL).

Some temporary threshold shift occurs at every SPL noted in figure 7–14, but shifts in excess of 15 db are the shifts which represent significant hearing damage, and these occur only with an SPL in excess of 85 db for normal exposure durations.

The interrelationships between noise exposure, NITTS, and NIPTS are, to a considerable degree, obscure. However, Glorig, Ward, and Nixon (1961) make the following statements about their relationship:

1. If there is no NITTS, there will be no NIPTS.
2. If the resting threshold is elevated, the magnitude of NITTS will be proportionately less.

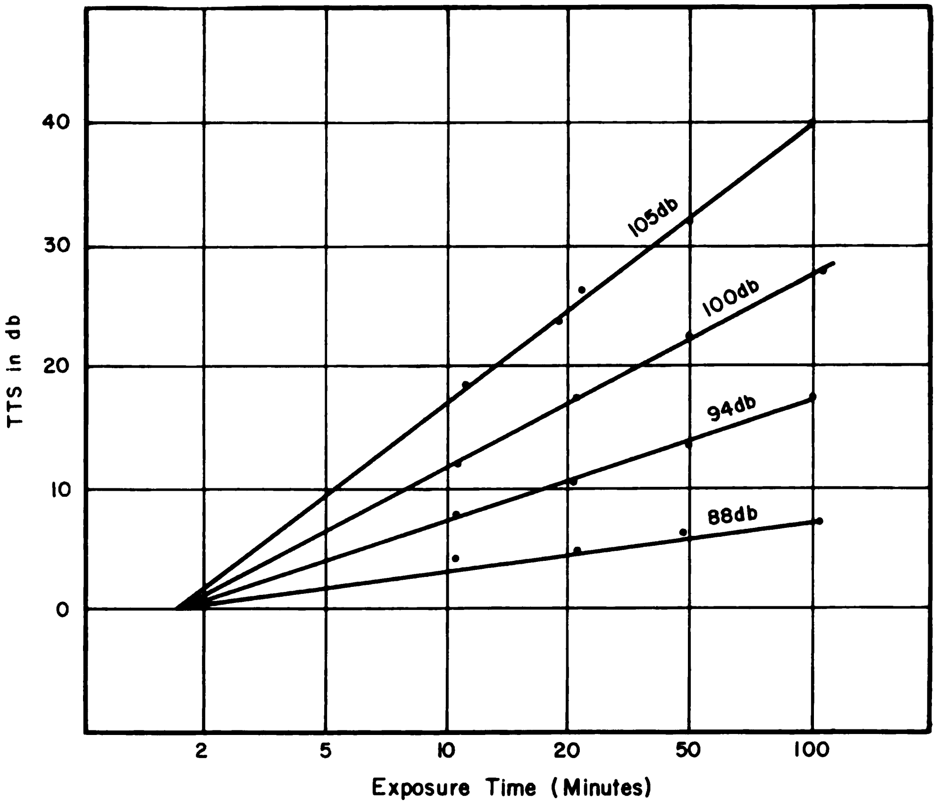

FIGURE 7-14. *Growth of TTS at 4 kc* (GLORIG, WARD, & NIXON, 1961).

3. A specific noise exposure (level and time combination) will produce a corresponding specific amount of NITTS.

4. A specific noise exposure (level and time combination) will produce a specific amount of NIPTS.

5. The progression of NIPTS is similar to that of NITTS, but with a different time scale.

These general rules are interesting in attempting to understand the nature of the hazard of noise, but more specific information about the subject is necessary.

Concerning temporary noise effects on hearing, Glorig (1961) states:

1. The temporary elevation of auditory threshold which results from one day's (8 hours) exposure to noise levels of 100 db or more may vary from none to a 35 db loss.

2. Exposure to a typical noise produces the largest NITTS at 4,000 to 6,000 Hz.
3. The major portion of the temporary loss is produced during the first one or two hours of exposure. (See fig. 7–14.)
4. The amount of NITTS is about the same for the same person from day to day, but it varies from person to person.

Concerning noise-induced permanent threshold shift (NIPTS) or permanent sensorineural deafness due to acoustic trauma, Glorig et al. (1961) state the following rules:

1. The maximum change due to noise exposure occurs within 10–12 years, regardless of exposure level.
2. The hearing level continues to shift upward after 10 years, but this shift parallels that due to "age-effect."
3. Each different noise level (time is constant) produces a specific shift.
4. At or below 80 db, there is only slightly greater NIPTS than that due to age-effect. (The age-effect curve is presented elsewhere in this chapter.)

TESTING AND FUNCTIONAL EXAMINATIONS[1]

The purpose of hearing tests is to determine the degree and type of any hearing loss and to estimate functional impairment as nearly as possible. Tests are useful in helping to diagnose certain diseases of the ear and to separate diseases of the sound-conduction apparatus from those of the sound-perception apparatus.

There are two types of hearing tests: simple (whispered voice, tuning forks) and audiometric (pure tone, speech discrimination). These are discussed below.

Simple Tests

Whispered Voice. "Auditory acuity shall be determined in the U.S. Navy by whispered voice tests only if audiometric equipment is not available. Individuals who are accepted into the Naval service upon the basis of the whispered voice test, shall be given a baseline audiogram upon arrival at the first equipped duty station" (chapter 15–10, *Manual of the Medical Department*).

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[1] This section is based in large part on the *Audiology* lecture notes of V. C. Bragg, Naval Aerospace Medical Institute. See "Bragg, 1967," reference at end of *Hearing* section.

Since audiometric testing alone is acceptable to BUMED for deciding upon acceptance or rejection of candidates, it will be discussed at length later. As a screening or indicative test, the whispered voice still has some value, but the results vary from examiner to examiner and from room to room. The technique of administering the whispered voice test is detailed in *MMD*. Prior to the testing, tympanic membranes and external auditory canals should be examined to insure that there is no occlusion of the external auditory canals by cerumen, which may result in a conductive type of deafness. Contrariwise, it must be remembered that otosclerosis and inner ear disease may cause profound deafness in spite of a seemingly normal tympanic membrane.

If the patient has sought out the examining doctor with a complaint of hearing loss, a very accurate history frequently will give a clue to the type of loss the patient has without any examination at all. For example, if the history discloses that the patient hears better in a noisy area, it is an indication of a conductive type of hearing loss, such as otosclerosis. If the history discloses better hearing in quiet surroundings, it strongly suggests a perceptive type of hearing loss.

Tuning Forks. Although tuning-fork tests are no longer included among Navy-approved examination procedures, they are useful clinically. For all practical purposes, the Flight Surgeon no longer needs a complete set of tuning forks. In less time than required to perform classical tests with all the forks, he can get much more information from audiometry. However, the *Weber, Rinne,* and *Schwabach* tests with a 512 fork are still useful and rapid, particularly for patients in bed. The 512 fork is selected because it is not felt as a vibration as in the case of 256 or 128, and higher frequencies are *heard* by air conduction (AC), not *felt* by bone conduction (BC). When sound is introduced into the skull by bone conduction, it is transmitted to both inner ears with very little loss regardless of where the sound source is applied. The *Schwabach* test will thus measure the hearing of the better ear only, unless one ear is masked. A simple improvised mask is a sheet of glazed paper rubbed rapidly over the ear to be masked. Since the *Rinne* test is a comparison of time of air conduction with bone conduction, masking requirements are the same as for *Schwabach*.

The *Weber* test is used solely to determine whether monaural deafness is of nervous or obstructive origin by comparing the bone conduction of the ears. If there is unilateral conductive deafness, the sound of the fork will be heard in the diseased ear; if there is unilateral perceptive deafness, the sound will be heard in the normal ear. This test is of value only if it corroborates other findings. It is performed by striking a fork of 512 double vibrations per second a maximum blow, damping the overtones and placing the stem firmly against the forehead or vertex and asking the patient, if he can differentiate, in which ear he perceives the sound. (Refer to figure 7–15.) The ***Weber*** test is not properly a test for hearing acuity but is useful solely for the purpose of determining whether the monaural hearing losses are, as noted previously, conductive or perceptive. As will be noted in figure 7–15, with the tuning fork resting on the vertex of the head, one part of the sound wave goes directly through the skull to the labyrinth. This is known as direct bone conduction. Another part of the wave goes through the skull to the tympanic cavity. This is known as indirect bone conduction. Part of this latter is reflected to the inner ear and part to the outside is lost. This happens normally on both sides, but if one ear is closed, one theory holds that the sound is heard much better on this side because the waves usually lost are reflected back, while on the other side they are lost. In lesions of the inner ear the perception of direct bone conduction is interfered with so that sounds are heard best on the unaffected side. Unfor-

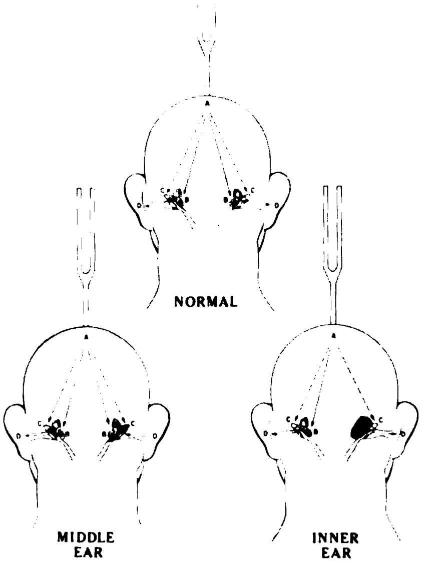

Figure 7–15. *Weber test.*

tunately, the classical responses noted above may not hold true because of the following:

1. Adults who have been hard of hearing for a long time from a unilateral conductive lesion will, at times, lateralize the sound to the side of the better-hearing ear.
2. The handle of any low-pitched tuning fork will vibrate with considerable amplitude. The patient may report that he hears the fork when in reality he feels it. The fork, therefore, should be one which will minimize this possibility of error (512 DV fork).
3. The sound of a fork of higher vibration (1,024 DV) is carried through the air so efficiently that the patient is unable to tell whether he hears it through the cranial bones or through the air. Therefore, it is preferable to employ a fork of lower pitch.
4. Some forks, when struck, produce overtones which may be louder than the fundamental tone of the fork, and the patient may hear the overtones through air conduction. This may be eliminated by using a fork which has no audible overtone and exercising care in stimulating the fork.
5. The handle of the fork must be pressed firmly against the head of the patient. If the handle is small, firm pressure may cause pain; therefore, the handle should be large enough to minimize this possibility.

The *Rinne* test is a comparison of bone conduction (BC) and air conduction (AC). Air conduction is normally longer than bone conduction. A medium-pitch fork (512) is placed on the mastoid until the patient states that he no longer hears it. It is then placed beside the tragus until he no longer hears it by air conduction either. When the air conduction time is greater than bone conduction, the *Rinne* is said to be positive. This is the normal condition. In middle ear disease, the air conduction is impeded so that hearing by bone is greater than by air (negative *Rinne*). In inner ear disease, bone conduction is impaired so that air conduction is increased over that of bone conduction. While this is also a positive *Rinne,* the ratio is far greater than that of a normal positive *Rinne.* (Refer to figure 7–16.) Frequently, in the recording of the *Rinne* test in order to avoid confusion over what positive and negative means, it is frequently recorded as bone conduction greater than air conduction, or, air conduction greater than bone conduction without making reference to positive or negative.

The *Schwabach* test is used to compare the bone conduction of the test subject with a normal (examiner). The fork is placed on the mastoid as in the *Rinne* test. Every few seconds the fork is lifted from the head and

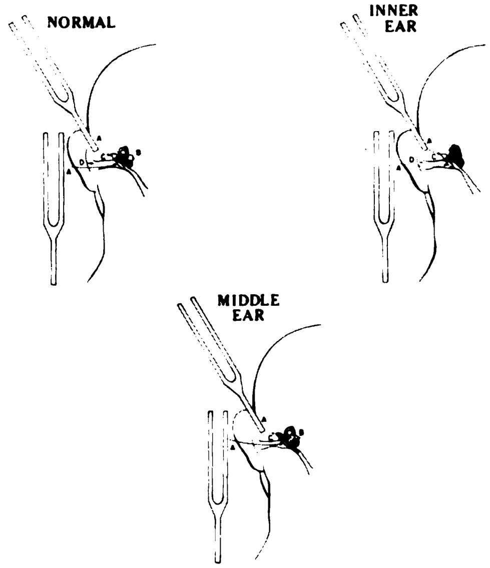

FIGURE 7–16. *Rinne test.*

then replaced in order to prevent "fatigue" of the nerve and permit a more reliable result. The observer may use his own conduction time or that of any young adult as a normal for comparison with the patient. By this method it is possible to determine roughly whether the subject's bone conduction is "normal," "shortened," or "very much shortened." In cases of middle ear disease, bone conduction is increased; consequently, the Schwabach is lengthened. In inner ear disease, the patient's bone conduction is reduced.

Malingering

People feigning deafness rarely claim bilateral hearing loss. Usually they insist that they have total loss of hearing on one side. Several tests have been devised to help detect the malingerer. The outstanding findings on any malingerer being tested are the inconsistencies of his reactions. It may be necessary to test a suspected malingerer repeatedly in order to verify positively the physician's suspicions.

The Stenger Test.—This test is done with two balanced forks of 512 double vibrations per second. For the uninitiated patient or test subject, the *Stenger* test is probably the most diagnostic for malingering. For the examiner, it is both the simplest and easiest test to administer (if it is understood). This classical test is given with the patient blindfolded and unaware that there are two forks, both being sounded at the same instant. The patient being examined is instructed to raise the hand on the side he hears the tuning fork or on the side of the ear in which he hears the sound. One fork is then held approximately 15 centimeters from the "hearing ear." The patient usually admits that he hears it. The second fork is then placed approximately 5 cm from the "deaf" ear. If the forks are of the same intensity, this closer sound will drown out (mask) perception of the first fork in the good ear. In actual unilateral deafness, the fork will always be heard by the unaffected ear at the same distance no matter where the opposite fork is held in relation to the "deaf" ear. In simulated unilateral deafness when one fork is held close to the "affected" ear, masking or drowning out of the sound coming into the other ear, the patient will be unable to state whether any sound is coming into the normal ear, and will automatically drop his arm or answer either late or incorrectly. With the fork close to the affected ear which he claims is deaf, the patient will indicate that he does not hear it, with the second fork 15 cm away from the normal ear, he will claim that he hears nothing if he is simulating. If the first fork is then taken completely away from the "deaf" ear, he immediately states that he hears the fork 15 centimeters away from the good ear; this proves that he would have heard the sound in the good ear if it had not been masked by the louder sound in the "deaf" ear.

The Lombard Test.—A second test for malingering is the *Lombard* test. A person with normal hearing will raise his voice when speaking or reading in a noisy atmosphere.

The patient is given easy reading material and requested to read out loud and continue no matter what happens. A Bárány noisemaker or tuning fork of rather loud intensity is then placed next to the supposedly good ear of the suspect while he continues to read. The patient who is truly deaf in the other ear will automatically raise the intensity of his voice as he continues to read, but the malingerer will continue to read in an even or very slightly elevated tone.

Audiometry

Modern diagnosis of deafness both in early stages and when it appears enough to be a handicap in an economic and social sense requires that the hearing be tested with the audiometer. The pure tone audiometer is designed to determine the acuity and, to some extent, the quality of hearing. It is an oscillator equipped to produce pure tones at a number of fixed frequencies at definitely measurable intensities.

The threshold of hearing is the minimum intensity at which a given signal is just perceptible. Many factors affect its measurement, so it must be considered in terms of a threshold measured with a particular signal and according to a particular technique. For example, the intensity at which a syllable is heard is not the same intensity at which a sentence may be perceived, or a given pure tone just heard. Likewise, the threshold for a continuous tone differs from that of a pulsed tone (interrupted tone), and each is different when measured when going from silence to the "just heard" point than from "hearing to not hearing." Therefore, to arrive at threshold measures which are comparable and repeatable, a standardized technique must be used.

It has been found that the most consistent pure tone thresholds are found using pulsed tones and increasing the intensity from silence to the "just heard" point. For purposes of standardization, the lowest point at which approximately half of the tonal presentations are heard is accepted as threshold. Thus, the definition of a pure tone threshold for these purposes is "the intensity (in decibels relative to average normal threshold) at which the ear under test is just able to detect the presence of an interrupted short-duration tone approximately 50 percent of the time, when the tone is presented by the ascending method." The ascending method indicates that the measure is made when increasing the tone from silence to the level at which it is just heard. "Approximately 50 percent of the time" simply means that if the test tone is presented to the ear, for example, five times, and the patient responds to three of those presentations, that level is accepted as the threshold point. Usually, two out of three correct responses are accepted as threshold.

Threshold is also measured "in decibels relative to average normal threshold." Thus, a threshold of 30 db at a given frequency means that this tone must be made 30 db more intense in order for the subject to hear it than it needs to be for the average normal person to hear it. It is then said that this person has a 30 decibel hearing loss at the test frequency. For this reason, the intensity control (attenuator) on most audiometers is labeled "Hearing Loss." A positive reading on the "hearing loss" dial indicates that threshold is higher (poorer) than normal, and a minus reading shows that the hearing threshold is lower (better) than normal. Standard procedure is outlined in figure 7–17. The following sequence should be utilized.

1. The patient is seated comfortably, facing away so that the examiner can see his profile but the patient cannot see either the examiner or the instrument.

2. Instructions are given simply but completely, like this: "I want you to listen very carefully during the test. You will hear a tone in one ear at a time. Sometimes the tone will be loud and sometimes very soft. Whenever you think you hear the tone, regardless of how loud or how soft it is, raise your index finger (right finger if the tone is in the right ear, left finger if the tone is in the left ear). Hold your finger up as long as the tone is on. As soon as you no longer hear the tone, drop your finger."

3. Next, the earphones are placed on the patient, red phone on the right ear and blue phone on the left ear. Care must be taken to insure that they are centered over the external auditory meatus.

4. The "output selector" of the audiometer is set for the right ear as a matter of standard procedure, unless the patient indicates that one ear is better than the other, in which case, the better ear is always tested first. The "tone interrupter" switch must be in the "normal-off" position so that the tone

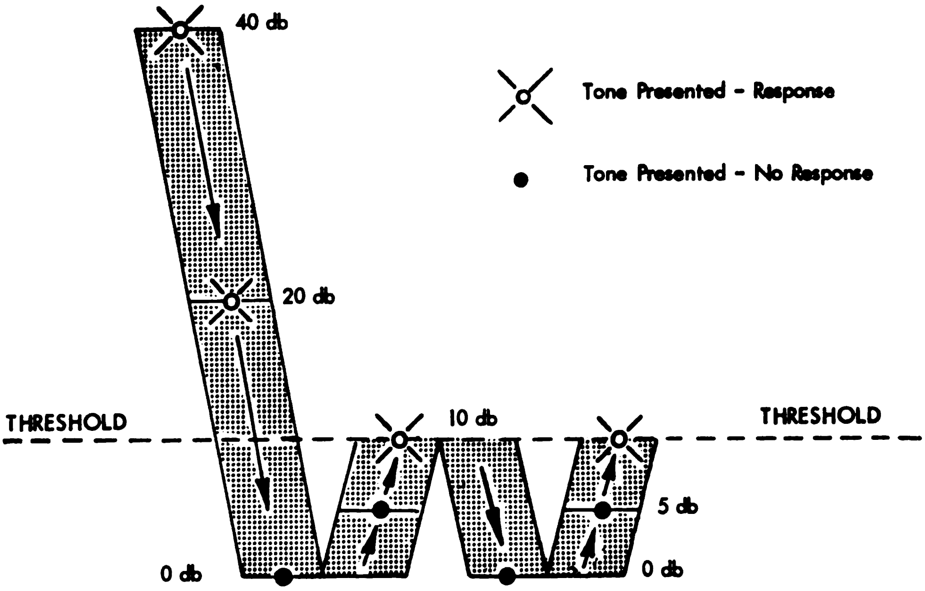

FIGURE 7–17. *Sequence of presentations used in establishing pure tone threshold. In example, threshold is 10 db. Patient responded each time tone equalled or exceeded this value.*

will sound only when you press the switch. The 1,000-cycle tone is always tested first because it is known to give the most consistently accurate results, so this is set on the "frequency-control" dial.

5. The "hearing loss" dial is set at 40 db, and the tone is presented by depressing the "tone interrupter" switch. If no response is obtained, the intensity is raised to 60 db and the tone is presented again. After the first response, the tone is dropped in 20 db steps for each subsequent presentation (varying the rhythm) until no response is obtained. The intensity is increased in 5 db steps until the subject again indicates that he hears the tone. Again, the intensity is dropped, this time by 10 or 15 db, and the ascent is started in 5 db steps until another response is obtained. Each time the patient hears the tone, the examiner drops down 10 or 15 db and again makes the 5-db-per-step ascent until a response is received. The lowest point at which two successive, or two out of three, correct responses are obtained is threshold for that tone and is so marked on the audiogram.

6. After finding threshold for the 1,000 cycle tone, the higher frequencies are tested in the same manner, and in ascending order (2,000, 3,000, 4,000, 6,000 and 8,000 cycles per second (Hz)). One thousand Hz may then be rechecked, followed by the low frequencies in descending order (500, 250, 125 Hz). Then the ear selector switch is turned to the opposite ear and the sequence is repeated.

Because the "hearing loss" dial may create unwanted transient sounds, it should never be moved while the tone is on. The "tone interrupter" switch must always be released before changing the hearing loss dial setting.

Masking.—It has already been indicated that the better ear is always tested first, but if there is no important difference between the ears, it is common practice to test the right ear first for the sake of standardization. There is an excellent reason for testing the better ear first, particularly when there is a difference of 40 db or more for air conduction (10 db for bone conduction) between ears. To explain the necessity for this, consider a patient who has one normal ear and one ear with a 60 db loss. If a test tone is presented to the poorer ear, it would have to be at a level of 60 db in order to be just heard. However, prior to reaching this level, the sound would be conducted through the skull and around the earphone cushions to the opposite ear which, being normal, would respond to the tone and lead the patient to indicate hearing at about 40 to 45 db. The examiner must be aware of this possibility so that he can prevent it by using a masking signal on the better ear to prevent its participation in the test of the poorer ear. However, since there is no way of telling how much difference there is between the two ears until they have both been tested, the better ear is tested first. The loss in the poorer ear will preclude its participation, so no masking is required. If the poorer ear were tested first, an erroneous audiogram would be obtained because of the participation of the better ear; and this ear would require retesting when it became known that the audiogram was in error. Testing the better ear first makes it possible to apply masking as soon as it appears that the poorer ear falls 40 db or more (10 db or more for bone conduction) below its partner, thus avoiding a complete retest of this (poorer) ear. The erroneous audiogram resulting from improper masking usually has a curve almost identical with the better ear, and falls about 40 db below it on the chart. This curve is called a "shadow curve" because it is actually a modified record of the hearing pattern of the better ear. Of course, masking should be used if the patient indicates that the signal sounds as though it were in the opposite ear, or elsewhere than in the ear under test. Whenever a test tone is heard in the ear not being tested, or elsewhere, it is said to be "lateralized."

Determining the proper amount of masking to use presents a serious problem. Although numerous systems of determining the proper level have been suggested, all require a knowledge of how much a given amount of the masking tone will shift the threshold for a particular pure tone. Since most audiometers are not calibrated to show this "effective masking level," a simple method which seems to give satisfactory results is used.

In air conduction testing, 60 db of effective masking has been found to be sufficient to rule out the better ear without being loud enough to interfere with measurements on the poorer ear. Since most audiometers have only 50–55 db of effective masking, the maximum setting is used when masking is necessary. With maximum masking on the good ear, if the tone continues to "lateralize" from the bad ear, the threshold is marked on the audiogram and a note is made that lateralization occurred.

Bone conduction testing is accomplished in the same manner as air conduction testing, except that the tone is delivered through the bone oscillator positioned behind the ear on the mastoid bone. Octave frequencies tested in this manner are 250 Hz to 4,000 Hz.

The following procedure is used in bone conduction testing, as it can be applied even though the masking calibration of the audiometer is not known. Assuming that the better ear has been tested and found normal, and the poorer ear has lateralized the tone, a given masking level with which to begin is arbitrarily selected. For example, 60 db on the masking attenuator dial may be selected, although there is no way of knowing what this may mean in terms of "effective masking." The bone conduction threshold of the poorer ear is then obtained while that level of masking is applied to the better ear. Next, the level of the masking is increased by 10 db and again the threshold on the poorer ear is obtained. If the thresholds are the same regardless of whether the masking control is set at 60 db or 70 db, the level of masking noise is clearly not interfering with the ear under test. If, on the other hand, the threshold should increase by 10 db when 70 db of masking is used, there may be two explanations: (1) 60 db of masking was insufficient, so that the threshold obtained with that amount of masking underestimated the amount of loss present; or (2) the increase in the level of masking noise caused an interference with the ear under test, and thresholds obtained with 70 db of masking overestimated the amount of loss. To decide which explanation is correct, the level of masking noise is increased and thresholds in the ear under test obtained at each 10 db increase of masking. If the threshold of the ear being tested increases proportionately with the increases in masking noise, it indicates that the masking noise is too intense, and was too intense at the beginning. When the proper level of masking is used, an increase or decrease of 10 db in the masking noise will not affect the thresholds in the ear under test. That is, the bone conduction thresholds in the ear under test should remain the same over a range of about 20 db in masking noise. The correct amount of masking must be determined in the manner described for each frequency tested, as the same level may not be suitable for all frequencies. It is important to remember that if masking is not used during bone conduction testing, the opposite ear must NOT be covered. Some bone conduction receivers are designed to fit into the headband in place of one of the earphones. In this case, it is important that the earphone not cover the nontest ear except when the masking is being used.

The Audiogram.—The form on which pure tone results are recorded is called an audiogram. The audiogram is a graph having two dimensions: Intensity along the ordinate and frequency along the abscissa. The intensity range goes from —20 to +100 db, and the frequency range from 125 to 8,000 Hz. The former is in decibels relative to average normal hearing.

Since the intensity (hearing loss) scale refers to average normal hearing, the zero (O) indicates no deviation from this norm. Any positive (plus) measure indicates a degree of "hearing loss," while a negative (minus) reading indicates a threshold which is better than normal. Thus, the farther down on the audiogram chart, the poorer the threshold, and the greater the intensity required to reach it.

The patient's threshold at each frequency tested is plotted on the audiogram for each ear separately both by air conduction and bone conduction. Standard symbols are used to indicate the threshold for each ear at the appropriate 5 db steps and separate symbols are used for air conduction and bone conduction thresholds. In addition, the right ear thresholds are drawn in red, while the left ear thresholds are indicated by blue symbols.

The symbols (O) and (X) are used to plot the threshold points at each frequency for the right and left ears respectively. ROUND, RIGHT, RED is a simple way to remember. Another memory aid is that the color coding is reversed from the Navy's running lights where the port light is red. The (O's) and (X's) which mark the threshold points across the audiogram are connected by solid lines of the appropriate color. Thus, at a glance, the contour of the hearing loss by air conduction can be seen for each ear. Bone conduction threshold points are connected by dashed lines.

The symbols for bone conduction thresholds are arrowheads (or brackets) pointing to the left or right. The right arrow would point to the patient's right ear if he were sitting facing the examiner. That is, the red arrow pointing to the examiner's left (patient's right) indicates bone conduction for the patient's right ear. There has been a great deal of confusion as to how these bone conduction thresholds should be recorded, but the foregoing system has now reached general acceptance.

Different symbols are used to indicate that the ear was tested while the opposite ear was masked. When masking is used, the (O) for the right ear is replaced by a triangle pointed up, the (X) by a square. The bone conduction symbols are changed from an arrow to a triangle by addition of a third line. Thus, if the right ear were tested by bone conduction while the left was masked, the symbol for the right ear would be a red triangle pointed towards the patient's right.

In clinical testing, 5 db increments are employed, even though finer measures could be made and some audiometers show one db steps. Therefore, the threshold symbols are placed on an intensity line or between two intensity lines, the former for even 10 db measures, the latter for 5 db steps.

Because the interpretation of the graph depends more upon its general configuration than its point-to-point variation, the dimensions of the chart itself are important. Standard audiogram forms show that the distance between octaves on the frequency chart (abscissa) is the same as that covering 20 db on the intensity dimension (ordinate). Adherence to this rule makes "reading" the audiogram less difficult than if various dimensions are used.

A note concerning "normal hearing." The zero reference line on the audiogram indicates normal hearing, but there is at present no universally accepted intensity which corresponds to this line. On the average, the British zero represents a sound pressure level 10 db less intense. This does not mean that in the United States people do not hear as well as in Britain, but only that each has accepted a different measure as its normal. In the United States, the results obtained from the 1935–1936 tests by the U.S. Department of Public Health are used. These results were based on tests of 1,242 persons having good hearing in both ears and no history of ear disease. The British standards were derived from tests on a few well-trained listeners, whose experience in the task accounts for the difference. In the near future, all will accept a new International Standard, which is much closer to the present British standard than to that of the United States. Therefore, a change of approximately 10 db in the United States "norm" can be expected.

The Pure Tone Audiometer.—The prototype of the modern vacuum tube and transistor-powered audiometer was developed in the 1920s. The Navy uses both manual and so-called "automatic" audiometers. The following comments (Bragg & Greene, 1963) apply to the manual type of instrument.

The audiometer is an instrument which generates tones of especially "pure" frequencies. That is, each tone contains a single frequency which is not contaminated by overtones or transient sounds. The intensity of these tones may be controlled in such a manner that it is possible for the examiner to adjust it to a level at which the subject is just able to detect the presence of the tone. This intensity may be read from the instrument, and indicates how much louder or softer this particular tone must be made for the subject than for the normal person to just hear it.

Only three controls are necessary for a manual audiometer, although most have additional switches and dials for convenience in presenting signals and for special tests. The required controls and their uses are:

1. *The Frequency Selector.* This control allows selection of any one of the test tones. Most audiometers have test frequencies in octave and half-octave steps from 125 Hz to 8,000 Hz. The older instruments had tones which were multiples of 64 Hz, but they are interchangeable with the more

modern test frequencies. Thus, 500 Hz and 512 Hz and 8,000 Hz and 8,192 Hz are so close to each other in frequency that either of the pair may be used for the other.

2. *The Attenuator.* The attenuator is also called the "hearing loss dial" or "hearing level control." Its action controls the intensity of the tone. Most audiometer attenuators are calibrated in 5 decibel steps, but some are accurate to 1 or 2 decibels. Zero decibels (0 db) at each frequency is average normal hearing. A positive (plus) reading indicates a hearing loss, while a minus reading means hearing which is better than the average normal. Most audiometers cover a range from minus 10 db to plus 100 db relative to this norm. A preceding section outlines the procedure which is to be used in determining threshold. This procedure must be followed exactly.

3. *The Tone Interrupter Switch.* This switch turns the tone either off or on, and "shapes" the tone in such a way that no unwanted "click" appears as it is turned on or off.

This switch is usually spring-loaded in the off position so that the tone is presented only so long as the examiner holds it on. It is important that the other controls (frequency selector and attenuator) be moved only when the tone is off. Movements of either of these controls while the tone is on may cause a click to be given with the test tone. Because such a click may contain frequencies other than the test tone, it would not then be known whether or not the subject heard the tone at the level indicated by the audiometer. His response might be to the click rather than to the tone.

These are the three essential controls. Most audiometers also have switches which allow each ear to be tested without changing the earphones, or for the test tone to be delivered through a bone conduction oscillator. Most also provide a "masking" tone which is used to drown out the hearing in one ear to insure that the opposite ear may be tested individually. Many audiometers have microphones and phonograph, and/or tape recorder inputs for use with speech tests. All of these features are extremely important, but are not used in the initial screening tests about which the Navy's Hearing Conservation Program is centered.

Automatic Audiometers.—In the Navy's Hearing Conservation Program two types of "automatic" audiometers are currently in use (Bragg, 1965). These are the Rudmose and the Tens instruments. The Rudmose instrument is available in either a single- or group-test form; the Tens audiometer is available only as a multiple-test device, testing up to 10 persons at a time.

Rudmose audiometers operate on principles first described by von Bekesy. A motor drives the attenuator and an attached marker, which records on an audiogram the movements of the attenuator. The subject holds a button which controls the direction of the attenuator's (and recorder's) motions. When the button is not depressed, the attenuator makes the tone increasingly louder; while the button is depressed, the tone becomes softer. The subject is told to press the button as soon as he hears the tone and to continue to hold it down as long as he can detect the presence of the tone, releasing it as soon as he can no longer hear the tone. In this manner, the recorder is made to move up and down across the subject's threshold, which is taken to be the point halfway between the recorder's excursions. Each frequency is tested in this manner for some 30 seconds, allowing sufficient time for a number of threshold crossings. Unless the threshold is crossed approximately five times in each direction, and these reversals are within 10 or 15 db of each other, the test should be repeated. Fewer crossings or a wider spread in the response pattern indicate a questionable threshold measure for that frequency. Such results often come about because of tinnitus, poor understanding of the test, or attempts to show a better threshold than one actually has. If it appears that insufficient threshold crossings will be accomplished within the allotted time, two procedures are available whereby the examiner may extend the time. The first of these is through use of the "delay switch." Moving this control to "delay" holds the audiogram tray for an additional 30 seconds. It should be used at the beginning of the test to give additional time for practice. The second method is to disengage the audiogram tray by holding down the attached lever. As long as the tray is thus disengaged from the gears, the subject continues to describe his threshold in the same manner, but the test time is not taken up. Both of these procedures allow the marker's excursions to be printed as a vertical line which can be interpreted at a later time.

One of the major testing problems in the Navy is that of determining the threshold of the person who wants very badly to pass a test, as, for example, that given as a part of a flight physical. Very often, such a person will establish a rhythm which gives a satisfactory pattern on the Rudmose audiometer, and continue pressing the button in this manner even though he no longer hears the tone. The audiometer provides an excellent means of detecting such persons and it should be used at least twice during the test. A description follows: The small plastic button protruding from the perforated front cover of the audiometer lowers the intensity of the test tone by 10 decibels so long as it is held in. When the subject has apparently established threshold for a given frequency, that is, when four or five crossings indicate the threshold level, the examiner should press the button. If the subject is actually responding to the test tone, it will be necessary for him to allow the tone to again reach threshold. That is, the indicated threshold will rise by 10 decibels. If he is not hearing the tone, but is responding to tinnitus, or is faking, the tracing will remain at the same level because he has no way of knowing that the level has dropped. As stated before, this procedure should be carried out at least twice on each test, and at any time that there is question as to the validity of a response. The recommended times for this are during the test for 3,000 Hz on each ear, and at other times when indicated by the results obtained. Stopping the table and changing the ribbon color at these times make the indications stand out clearly so as to preclude later misreading.

The Tens audiometer tests hearing by presenting a series of pulsed tones at various levels. The subject counts the pulses heard while a light before him signals "Listen," then presses the button marked with that number when the light signals "Respond." A correct response causes the audiometer to make a mark in the appropriate space on the audiogram, and the lowest intensity at which such a mark appears is the threshold for that tone. Ten subjects are tested at one time, and their responses are automatically recorded on the ten audiogram forms.

SPEECH AUDIOMETRY[2]

The signal/noise ratio is one of the factors which affect speech intelligibility. The absolute intensity (loudness) of meaningful sound (speech, in this instance) is not nearly so important as the loudness of speech in relation to the loudness of the background noise. The speech/noise loudness ratio (signal/noise ratio in electronics) determines whether speech is intelligible at all in many military applications. For example, with a background noise level of 100 db, one must have a 110 db level of speech for better understanding. However, the human being can only manage about 85 db from the unaided larynx. Therefore, without artificial amplification, speech would be completely unintelligible.

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[2] See "Bragg and Greene, 1963," reference at end of *Hearing* section.

A speaker monitors his own voice continually. If the sound of his own voice is delayed in reaching his ear, speech becomes difficult, if not impossible.

Unilateral deafness is more a handicap than a disability. It causes difficulty in localizing the source of a sound, and when the source is on the deaf side, the head casts a "sound shadow" which interferes with high frequencies, thus making the consonants of speech difficult to hear. As compared with binaural hearing, loudness is decreased by about 5 db. Industrially, deafness in one ear is considered a 10 percent disability if the other ear is normal.

Certain frequencies are more important in the interpretation of speech than others. The most important frequencies are the 500, 1,000, 2,000, and 3,000 Hz. Speech is essentially compressed into this range, but these frequencies are sufficient for fairly complete understanding.

Vowels generally have greater intensity and have most of their energy in the frequencies below 1,000 Hz; consonants have less intensity and most of their energy is in frequencies above 1,000 Hz. The frequencies were arbitrarily graded according to speech importance: 2,000 Hz would be most important and have an assigned value of 40 percent; 1,000 Hz would be next with 30 percent; and 500 and 3,000 to 4,000 would each receive a value of 15 percent. Since consonants are necessary for the recognition of words, especially out of context, intelligibility of speech is usually affected by moderate or severe high tone loss.

Many attempts have been made to assess hearing loss for speech from pure tone audiograms. When the hearing loss for speech frequencies produces a fairly flat curve, good correlation between average loss by audiogram and measured speech reception exists. When the loss is sensorineural, with abrupt changes between octaves, great disparity often exists between the average of these three speech frequencies and measured speech reception. In patients whose audiogram curves exhibit an abrupt drop, the average of the best two frequencies may give better correlation. The apparently good speech reception threshold in patients with an abrupt-drop audiogram curve may give an optimistic view of the disability for speech. Discrimination is usually bad when the drop affects speech frequencies. (This is the patient who says, "I can hear you but I do not understand you.")

Speech is commonly heard at intensities well above threshold (30 db), and this factor should be given considerable weight in assessing hearing loss. With the advent of the speech audiometer, the threshold for speech can now be measured under clinical conditions, often with greater precision than for pure tones. Speech audiometry is now primarily given either by live voice or tape-recorded. A threshold of speech similar to a pure tone threshold is determined. This threshold is determined by the use of what is known as spondee words. These are two syllable words with equal stress, such as railroad, oatmeal, and hotdog. A satisfactory technique is to establish rapidly an area between no response and complete response and then determine the level at which 50 percent of the words are repeated correctly by the patient being examined. When the threshold has been established, it usually will range very closely to the average of the so-called "speech frequencies" (500, 1,000, and 2,000 Hz) by pure tone audiometry. After the determination of threshold speech audiometry, the ability to discriminate is tested by the use of more difficult phonetically balanced (PB) monosyllabic words; such as, kick, muss, purge. Patients with normal hearing and patients with conductive deafness usually prefer to listen to speech at a level of from 30 to 35 db above the spondee threshold. Patients with a so-called recruiting sensorineural deafness usually prefer to listen at levels less than this. With the use of the PB words, the normal patient and the patient with a pure conductive loss will have a percentage score of the words of somewhere between 90 and 100 percent reception. It is said that patients with what is known as a mixed-type deafness, deafness of both conductive and sensorineural nature, will usually receive a score of somewhere between 80 and 90. Patients with a pure sensorineural-type loss will usually have a score below 80 percent reception. The routine inclusion of discrimination studies in all cases of perceptive deafness has made it possible to predict with considerable accuracy whether or not a patient will have difficulty adjusting to a hearing aid or will require lip reading for adequate communication. Threshold audiometry is equivalent to measuring the light sense in the eye. Discrimination studies are comparable to the Snellen chart readings. With the advent of audiometric testing, in addition to the pure tone and speech reception testing, a combination of these types of testing has been added which is known as recruitment.

Recruitment is defined as an abnormal increase in loudness as intensity increases. In the European literature, this is referred to as regression, in that the hearing loss is decreased as the intensity of a stimulus is increased. In patients with recruitment, the discrimination score is characteristically poor and may decrease with increased intensity well above the threshold. The findings are those of "compressed" hearing in that the patient fails to hear low-intensity sounds and reaches the discomfort level when intensity is only moderately increased above his threshold. Often discomfort is experienced at an intensity that is not unpleasant to a normal ear. This explains the familiar sequence: "What did you say?" With the repetition at a slightly greater intensity, the response may be, "Don't shout at me!" Tinnitus, often of a loud, hissing type, may be the most distressing symptom.

In order to determine the true state of hearing, a single examination is seldom adequate. Wide fluctuations of hearing on different days should suggest either fluid in the middle ear, end organ disease, or functional deafness. Before any therapy is undertaken for chronic deafness, it is necessary to have the results of at least two audiometric tests, preferably at different times and different days, that agree within ± 5 db for each frequency, before one can assume that this represents the true threshold. By means of hearing tests, both the location of the lesion causing impairment of hearing and the degree of impairment can be established. From a diagnostic standpoint, the aim is to be able to state the degree of involvement of the conductive mechanism, the inner ear, exclusive of ganglion cells, and of the cochlear nerve. The conductive mechanism includes lesions of the external ear, middle ear, and the Eustachian tube. The two types of middle ear lesions should be distinguished: Lesions which produce increased mass and those which produce increased stiffness. Lesions involving the round window, such as adhesions connecting the round and oval window (short circuit), and drum perforations also cause deafness by interfering with "phase" relationships. Lesions causing increased "mass," such as exudative lesions, characteristically produce an air conduction loss which is greater for high frequencies than low; the reverse is true for lesions causing increased stiffness, such as otosclerosis. This is in accord with the physical laws of impedance. Most cases of sensorineural deafness are characterized by a predominant high tone loss which is equal for both bone and air conduction. The limit of bone conduction measureable by most audiometers is considerably less than that measureable by air conduction. When air conduction loss exceeds this limit, no bone conduction measurement should be obtainable at that frequency. This is usually recorded by indicating the limit tested with an arrow pointing downward. When there is complete or nearly complete loss demonstrable by air conduction, there is usually residual hearing by bone conduction for at least 250 Hz and usually 500 Hz in the range of about 35 to 40 db for 250 Hz and 45 to 55 db for 500 Hz. These findings are probably due to vibration and should not be interpreted as indicative of a mixed deafness at these frequencies. Discrimination scores in sensorineural lesions vary with the degree of involvement; thus, even with profound losses restricted to the higher frequencies (4,000 and up), discrimination may be relatively unaffected. When the speech frequencies become involved, discrimination becomes increasingly impaired. In pure sensorineural deafness, the discrimination score characteristically increases with intensity above threshold to a plateau, and true discomfort is usually absent.

The Naval Aviator's Speech Discrimination Test (NASDT) (Bragg & Greene, 1963) is a test (utilizing a speech audiometer) designed to determine the aviator's ability to understand speech signals in aircraft noise backgrounds. The test uses a standard phonetically balanced (PB) wordlist containing all the speech sounds which are found in the English language and in the proper relationship with each other. The words were recorded with a background of C–45 cockpit noise. The Sound Pressure Level (SPL) of the noise is 100 db, approximating that in C–45 cockpits in level flight. The SPL of the words is 115 db, a level chosen by a panel of listeners as optimal in this noise background.

It has been found that there is no consistent relationship between NASDT results and those of pure tone audiometry. That is, some aviators who show marked hearing loss on pure tone audiometry show no loss in their ability to hear words in a simulated cockpit situation. Occasions for administration of the NASDT are promulgated by current directives.

Instruments used to administer the NASDT are located at the following locations, and Flight Surgeons should refer naval aviators requiring the NASDT to the closest station:

Naval Missile Center
Point Mugu, Calif.

Marine Corps Air Station
El Toro, Santa Ana, Calif.

Marine Corps Air Station
Cherry Point, N.C.

Naval Air Station
North Island
San Diego, Calif

Naval Air Station
Quonset Point, R.I.

Naval Air Station
Oceana
Virginia Beach, Va.

Naval Air Station
Cecil Field, Fla.

Naval Air Station
Key West, Fla.

Naval Air Station
Albany, Ga.

Naval Air Station
Glenview, Ill.

Naval Auxiliary Air Station
Ream Field
Imperial Beach, Calif.

Naval Air Station
Miramar, Calif.

Naval Air Station
Lemoore, Calif.

Naval Air Station
Whidbey Island
Oak Harbor, Wash.

Naval Air Station
Norfolk, Va.

Naval Air Station
Barbers Point, Hawaii

Naval Air Facility
Washington, D.C.

Naval Aerospace Medical Institute
Naval Aerospace Medical Center
Pensacola, Fla.

Psychogalvanometry

Psychogalvanometry is a last and useful adjunct in the determination of certain types of deafness. This test is based on the fact that when a subject is shocked or otherwise emotionally stimulated, he reflexly secretes perspiration. If the resistance between two electrode points, on the hand, is included in a wheatstone-bridge circuit and the bridge is balanced, this balance is upset whenever there is a change in moisture under the electrodes. Thus, if the patient reacts to an electric shock by perspiring, change in resistance is recorded as a sudden change in the balance. The shock should be of sufficient intensity to be unpleasant but not painful. When it is noted that constant responses are obtained from the shock, the patient is given a loud pure tone warning before delivering the shock. This is repeated until the patient associates tone and shock and responds to the tone in the same manner as if he had been shocked. He is now conditioned, and the intensity of the sound can often be lowered to threshold, thus obtaining an objective audiogram. Results of this test are valid if distinctly positive, but negative results may be due to inability to condition the patient or to technical difficulties. In other words, we may be able to state that the patient has hearing of at least so much but we cannot say that he does not hear at certain intensities. The test is much easier to perform and more reliable on adults and is useful to verify results obtained by subjective audiometry in cases of functional deafness (conversion states and malingering).

Interpretation of Audiograms.—When hearing loss is graphically presented on an audiogram chart, the following descriptive terms are often used to describe the shapes of the curve:

Flat. Same loss at all frequencies.
Ascending. Greater loss in low frequencies.
Descending. Greater loss for high frequencies.
Abrupt Drop. A change of 40 db or more in one octave.
Basin. Greatest loss in middle frequencies.
Dip. A depression and subsequent return to former level.
Peak. An elevation and subsequent return to former level.

Certain factors affect the audiogram in various ways. Conductive losses such as otitis media, otosclerosis, etc., may depress the entire audiogram, often with greatest effect in the low frequencies, yet the bone conduction curve little or none. Improper placement of the earphones usually affects the low frequency portion of the air conduction curve more than the high frequency region. A similar effect may be caused by insufficient practice on some automatic audiometers before beginning the test, and by excessive ambient noise. The latter also affects the bone conduction curve. Thus, a "rising" threshold, in which the lower frequencies are depressed more than the 1,000, Hz tone, may be an indication of poor testing or of a conductive loss which does not affect susceptibility. If careful testing reveals that a true hearing loss exists, it should be treated otologically. Unless a conductive loss precludes the wearing of ear protection, it does not contraindicate work in noisy areas.

A "flat" threshold curve in which all frequencies are depressed approximately the same amount may be caused by poor understanding of the test by the patient (he simply waits until the tone is comfortably loud rather than responding at threshold) or by certain conductive losses. A flat audiogram may also result from Ménière's disease. This is a sensorineural loss and must be considered as an indication of susceptibility to noise damage. The Ménière's case can be distinguished from the flat conductive curve by a similar depression in bone conduction, and by a history of vertiginous attacks with nausea and vomiting. Recruitment tests are always positive. Where bone conduction testing is not available, the *Rinne, Schwabach,* and *Weber* tests may be used to differentiate the conductive loss from the sensorineural. These tests are explained elsewhere.

The step-by-step procedure for evaluating audiograms follows:

1. First, the three speech frequencies (500, 1,000, 2,000 Hz) are considered. A loss in this region in excess of 20 to 25 db indicates that the hearing for speech may be affected (if the test is accurate). A "hearing loss" of 15 db or less is considered within normal limits. If the average loss for these three frequencies is 30 db or greater, the patient should be experiencing difficulty in everyday conversation, and is in need of otological and/or audiological help.

2. Next, the overall level and configuration of the audiogram are noted. If the curve is depressed throughout the test range and the otoscopic examination negative, it is possible the patient is not responding at his true threshold. He should be carefully retested, with instructions to respond when he is *just barely able* to hear the tone. Of course, any medical problem found should be treated before retesting. If such a problem prevents the wearing of proper noise protection equipment, the patient should be kept away from noise until his difficulty is cleared up.

3. Next, the lower frequencies (below 1,000 Hz) are observed. If this portion of the audiogram is "rising" (500 Hz lower than 1,000, 250 lower than 500, etc.), the following possibilities must be considered:

a. Was there too much ambient noise in the test room?

b. Does the patient have a conductive hearing loss (otitis media, blocked Eustachian tubes)?

c. Were the earphones improperly fitted?

d. Did the patient have sufficient practice on the automatic audiometer?

None of these factors should affect the patient's ability to work in loud noise, except as indicated above.

4. Finally, the high frequencies (those above 1,000 Hz) are considered. If this portion of the audiogram "falls" (poorer hearing with increased frequency), the patient may have a temporary or permanent threshold shift. In interpreting such cases, two factors should be remembered.

The *first* is that while losses from noise exposure first appear on an individual's audiogram in the region of 4,000 Hz, hearing loss through aging also shows up in this region. Thus, allowance for the aging effect on hearing must be made.

Figure 7–18 illustrates the differences in hearing acuity for man that occur through advancing age. The chart shows, for example, an average hearing loss of approximately 12 db at 4,000 Hz at age 40. It can be seen that, with

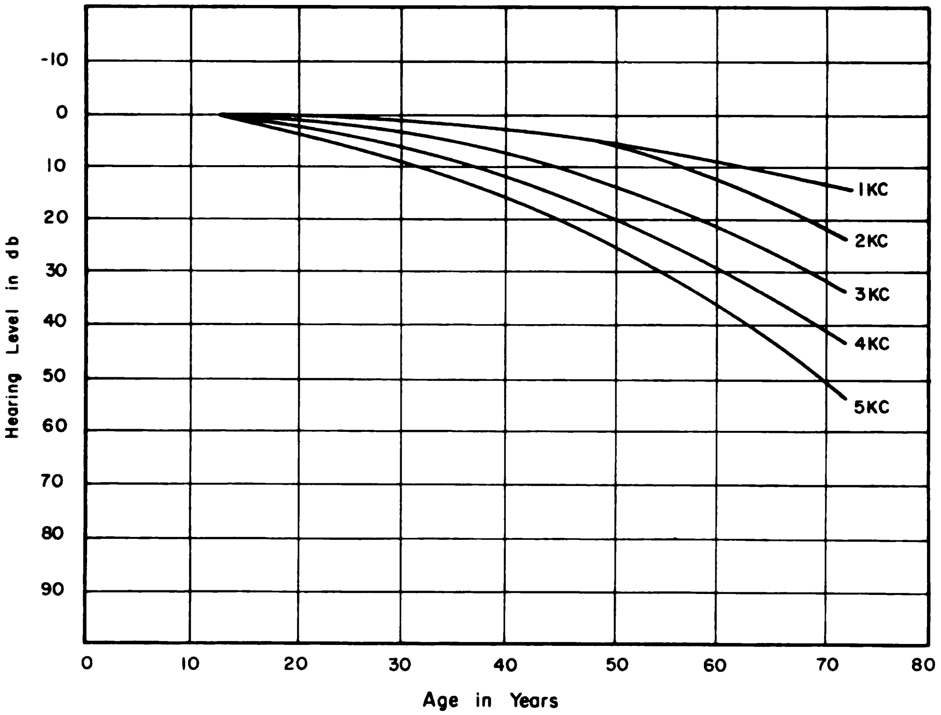

FIGURE 7–18. *Hearing loss as a function of age—nonoise occupation* (GLORIG, WARD, & NIXON, 1961).

age, hearing loss (permanent threshold shift) is greatest in the higher frequencies.

The *second* is the TTS. The temporary hearing loss induced by a single day's exposure to high noise levels can approximate 35 to 40 db. It is therefore quite important to realize that audiometry performed at the end of the day may be quite misleading in determining permanent hearing loss.

The hearing loss which results from long exposure to high-intensity noise is usually reflected in the audiogram by the occurrence of greatest loss around 4,000 to 6,000 Hz, with perhaps some recovery at higher frequencies. Because of this configuration, and its common occurrence among aviation personnel, it is often referred to as "aviators' notch." The loss is sensorineural in nature, and equally affects both air conduction and bone conduction curves. Typically, the loss begins at 4,000 or 6,000 Hz, increasing and spreading to the higher and lower frequencies with continued exposure. Thus, the noise-damaged ear can be recognized by the audiogram in which hearing acuity is progressively worse between 1,000 and 4,000 Hz (sometimes 6,000 Hz).

THE MILITARY AVIATION NOISE ENVIRONMENT

As a first step toward assuring personnel protection, the Flight Surgeon should acquaint himself with the noise situation on his air station or ship. This will allow him to make meaningful recommendations in the event certain changes in ground operations appear warranted and feasible. For example, he may recommend a less inhabited area for engine runups. A knowledge of the noise hazard areas is also essential in order to assure that personnel who must be in these locations are equipped with and are in fact using appropriate ear protection devices.

Noise in Flight

The ambient noise environment for the aircrew and passengers in flight varies somewhat among jet-powered aircraft, turboprop-powered aircraft, reciprocating-engine aircraft, and helicopters. The following briefly describes the characteristics of each of these aircraft noise environments.

Jet Aircraft.—Noise within turbojet- and turbofan-powered aircraft is produced primarily by the engines and by aerodynamic disturbances. Both result in a continuous noise spectrum in which every audible frequency is represented, with little variation in intensity among frequencies. Such noise is generally referred to as "white" noise. During ground operations and the initial stage of takeoff, the engine is the main contributing factor. With increased airspeed, the aerodynamic noise becomes more prominent and can equal that of the engine.

In figure 7–19, it can be seen that, depending upon the particular power setting, overall sound pressure levels were in the range 111.5 to 113 db. Most high-speed jet aircraft have similar noise curves, although the overall levels may be slightly lower or higher.

For multiengine jet aircraft, noise levels vary at the different crew positions. In one instance the noise level at the pilot's position was measured at 100 to 110 db in level flight while at the navigator's position it ranged from 92 to 98 db. In another aircraft the cockpit level ranged from 86 db during taxi to 100 db during climb, while the overall level in an aft crew position was 108 db (data taken from O'Connell, 1960). Noise levels are somewhat lower at crew stations forward of the engines.

Fixed-Wing Propeller Aircraft.—Fixed-wing propeller aircraft' generate intense low-frequency noise within the cockpit and cabin, caused

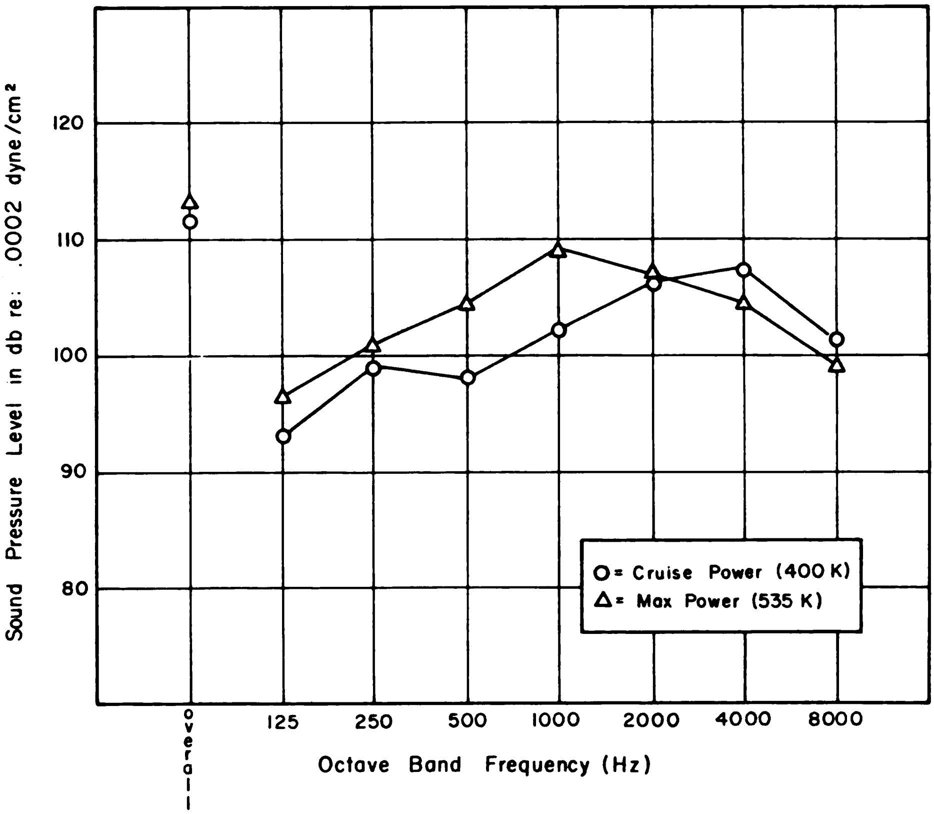

FIGURE 7–19. *Noise spectrum in cockpit of A-7A aircraft at 3,000 feet—air refueling probe faired* (HARRIS & MUSSON, 1966).

mostly by the rotating propeller tips. The fundamental frequency of propeller noise is usually less than 200 Hz. Noises from the engines and from fuselage vibrations are similar to that of the propeller tips but are of less intensity. Although aerodynamic noise is still present, it is of low intensity and almost unnoticeable.

Noise levels in aircraft of this type range from 90 db to as high as 130 db as a function both of type of aircraft and conditions of operation, being loudest during preflight check, takeoff, and climb. Noise levels are most intense in the vicinity of the propeller and in multiengine aricraft vary considerably with crew position.

In turboprop-powered aircraft the noise characteristics are much the same as in conventional reciprocating-engine aircraft although there is some higher frequency noise generated by the engine.

Helicopters.—Noise levels and vibration within many helicopters can be very intense. The major sources are the power plants, transmission and shaft-distribution units, engine exhausts, and rotor and antitorque systems. Figure 7-20 shows noise levels measured at the pilot's position in various helicopters.

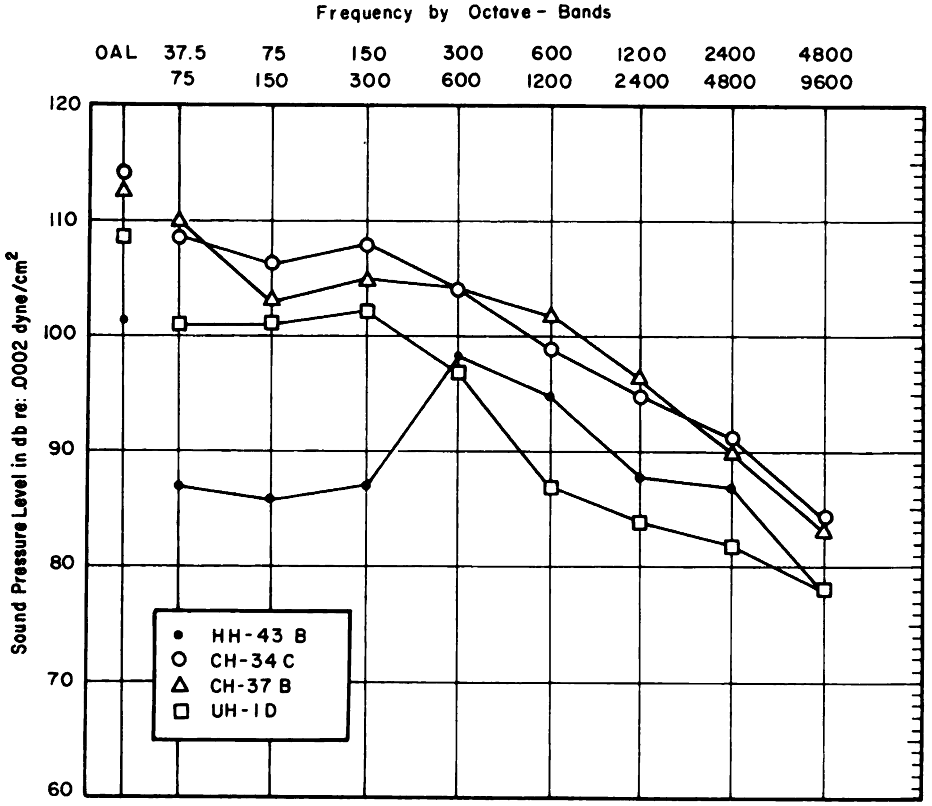

Figure 7-20. *Noise exposures at pilot position in various helicopters during normal cruise* (Gasaway, 1964).

With larger helicopters, noise has become of serious concern not only at crew stations but also in the passenger compartment. An investigation of the noise problem in the CH-37C (a twin-engine Marine Corps assault helicopter) showed that the passenger compartment measured between 114 and 122 db in flight with an average of about 119 db, representing an increase of

5 db in the overall noise level in the passenger compartment over the CH-19E, previously used as a troop carrier. The pilot's area of the CH-37C registered a level of 108 db.

A frequency analysis of the noise showed small peaks between 70 and 1,200 Hz with the rotor turning on the ground but little noise above 2,000 Hz. During taxi and in flight there were several very predominant peaks in the low frequencies and one from 2,100 to 2,200 Hz.

Noise on the Ground

The environments in which aviation personnel must carry out their duties on the ground, particularly on the carrier flight deck, now include some of the most intense noise levels associated with aircraft operations. The contributors to ground level noise are many and varied and include such aircraft activities as startup and taxiing, pretakeoff runups, takeoff including afterburner employment, maintenance runups, and aircraft flyovers. Ground power units create significant noise levels. Also, these noises can impinge upon the hearing of a large number of people at any one time. Maintenance personnel, plane directors and handlers, the catapult crew, and the many other personnel who are stationed on or near the flight deck/flightline during air operations and maintenance activities, can become completely immersed in a field of extremely high-intensity, high-frequency, and therefore potentially injurious noise.

Jet Aircraft.—Probably the most harmful noise-generating source is the jet engine, both turbojet and turbofan. The noise produced by such engines is of the broad-band type but contains narrow-band high-frequency components throughout the major power range of the engine.

The best way to illustrate the extent of the jet noise problem for ground personnel is to present the results of a number of surveys of naval aircraft noise emission conducted by the Service Test Division, Naval Air Test Center, Patuxent River, Md., under project No. PTR PP-3675.

The purpose of the tests described in the report of 20 October 1961 was to provide specific sound measurements of various turbojet aircraft with their engines operating at power approach thrust. The measurements were made at a distance of 100 feet from the outlet of the tailpipe at a relative bearing to the aircraft of 150 degrees. The results are presented in table 7-2.

TABLE 7-2

Sound Intensity Levels for Various Turbojet-Powered Aircraft Operating at Power Approach Thrust

(Measured at point 100 ft from the outlet of the tailpipe at a relative bearing of 150°)

Aircraft	Rpm percent SPL (db re.0002 dynes/cm)[2]	SPL (db re.0002 dynes/cm)[1]
A-4C (A4D-2N)	83	110
F-6A (F4D-1)	86	115
F-8D (F8U-2N)	86	117
F-4A (F4H-1F)	85	123
EA-3B (A3D-2Q)	90	125
A-5A (A3J-1)	83	128

[1] Both engines.

The purpose of the survey described in the report of 15 April 1963 under the referenced project was to determine the engine noise profile of the A-4E aircraft. Measurements were made at various distances up to 200 feet from the tailpipe with the engine operating at military power. The maximum overall noise level encountered was 152 db on the 12.5 foot semicircle at the 150 degree position. The sound level readings for other locations are presented in table 7-3.

TABLE 7-3

Angular Distribution of Sound Pressure Level in the A-4E Airplane at Military Power

Distance from aircraft (circle radius, feet)	0°	30°	60°	90°	120°	150°
12.5	141	138	139	140	143	152
25	131	130	132	134	140	151
50	121	123	126	129	136	143
100	117	118	121	125	134	129
200	112	116	115	117	128	114

TABLE 7–4

Frequency and Angular Distribution of Sound Pressure Levels from A–4E Aircraft at Military Power

Frequency (Hz)	0°	30°	60°	90°	120°	150°
Overall SPL	141	138	139	140	143	152
40	104	101	105	104	111	118
50	104	103	105	106	113	123
64	105	106	106	107	111	125
80	101	98	101	103	107	116
100	107	105	110	110	114	126
125	109	108	110	112	116	126
160	114	112	112	113	121	134
200	115	114	113	114	126	138
250	115	114	113	115	127	139
320	116	116	115	119	124	140
400	122	119	119	122	126	142
500	134	123	126	123	130	143
640	135	132	133	128	131	143
800	133	130	133	134	131	141
1,000	130	128	130	134	134	140
1,250	128	126	129	129	133	137
1,600	125	124	127	129	133	135
2,000	124	121	125	128	133	133
2,500	122	122	125	127	133	132
3,200	120	119	123	125	131	130
4,000	117	115	121	124	129	127
5,000	114	113	118	122	124	123
6,400	112	111	114	119	121	120
8,000	102	102	104	107	110	110
10,000	101	109	104	108	109	108

NOTE.—Measured at 12.5-foot radius.

Table 7–4 presents a breakdown of the sound pressure levels at various locations by frequency. Similar engine noise profiles were determined for a number of other Navy jet aircraft (see *References*). Table 7–5 presents some of the maximum overall noise levels obtained.

As another part of the NATC project, tests were conducted to determine the sound levels to which flight deck personnel would be exposed during catapult launching of the F–4B and A–5A aircraft.

It was found that during military power launches of the F–4B, personnel on the flight deck and in the catwalks adjacent to the aircraft are subjected to noise levels of 138 db for 10 to 15 seconds, and momentarily to 142 db as the aircraft is launched. For combat power (afterburner) launches, the levels are about the same, with a momentary exposure of 152 db. These

TABLE 7–5

Maximum Overall External Noise Levels From Navy Jet Aircraft

Aircraft	Power setting (both engines)	Overall noise level (db)
F–4B (F4H–1)	Military	151
	Maximum with afterburner	156
A–6A	Military	156
A–5A (A3J–1)	Military	152
	Maximum with afterburner	156
A–3A (A3D–1)	Military	155

readings do not represent the maximum levels generated by the aircraft but are considered representative of the noise levels to which flight deck personnel are exposed.

The report also notes that aircraft passing over the deck after waveoff may produce noise levels as high as those reached during launching.

The measures made of the A–5A were slightly lower but did not differ significantly from those of the F–4B.

Fixed-Wing Propeller Aircraft.—In this type of aircraft, the primary noise component is generated by the propellers, but when the engine is operating at low rpm, some noise from the exhaust is evident.

Figure 7–21 indicates the noise characteristics at various angular positions 200 feet from a large multiengine cargo aircraft.

Helicopters.—The most important noise created by reciprocating-engine helicopters is that generated by exhausts, rotors, and antitorque systems (Gasaway, 1964). Figure 7–22 shows the measures obtained beneath and ten feet to the side of the exhaust ports of one engine of a large twin-engine helicopter while the engine was operating at an idle of 1,500 rpm and 16 inches of manifold pressure. The rotors were not engaged.

Power Units.—Another source of noise to be considered is the ground power unit. This may be an auxiliary electrical power unit, a gas turbine unit for providing compressed air, or an air-conditioning unit. Such equipment can generate very high noise intensities which, over extended periods of use, can be as harmful to the hearing as the noise from aircraft engines.

Other Sources.—Final mention should be made of the fact that the ground level noise problem is not restricted to the flightline or the flight deck.

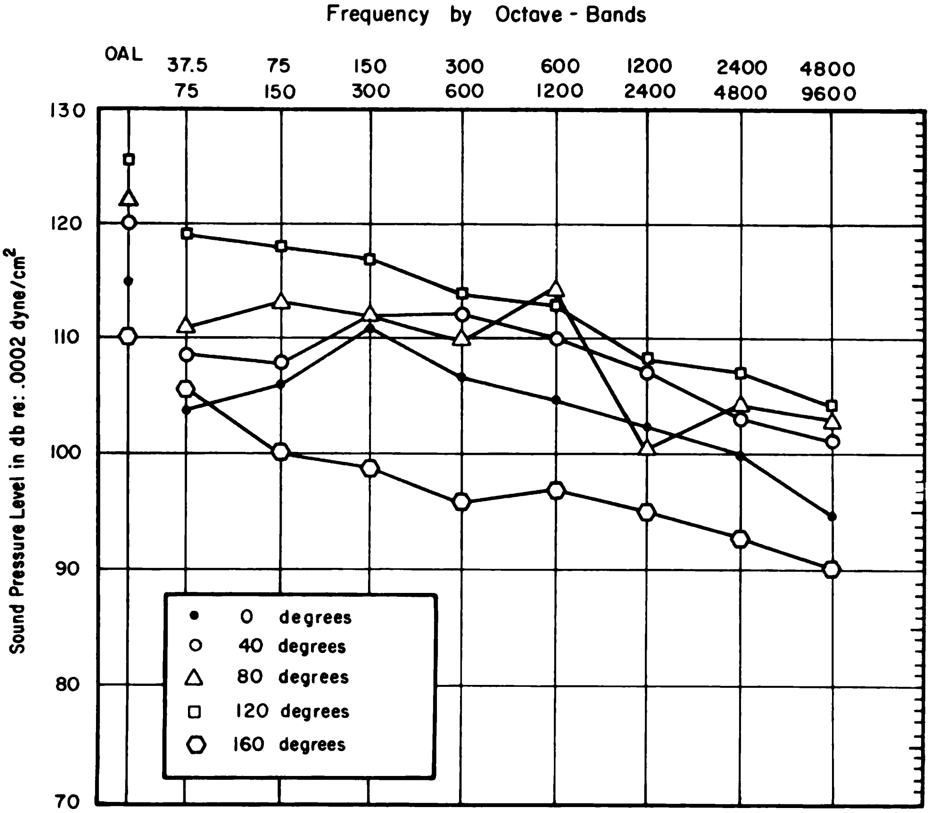

FIGURE 7-21. *Noise generated at various angular positions 200 feet from a large multi-engine cargo aircraft* (GASAWAY, 1964).

Ashore, noise impinges upon the hearing of personnel working in nearby hangar areas and offices. The day-to-day noises experienced by personnel on an aircraft carrier while it is underway and carrying out its schedule of flight operations provide the most striking example of this fact. Personnel working, sleeping, or just passing through compartments located below the catapults and the runway, in the vicinity of elevator machinery rooms, the steering gear room, and inside the lower areas of the island, can be exposed to noise levels in excess of 100 db without ever setting foot on the flight deck.

In addition, ground personnel at shore stations may have off-duty employment in noise hazard jobs of which the Flight Surgeon is unaware. In some cases, recreational activities, such as automobile racing, engine tuning of nonmufflered engines, or model aircraft racing produce severe noise hazards.

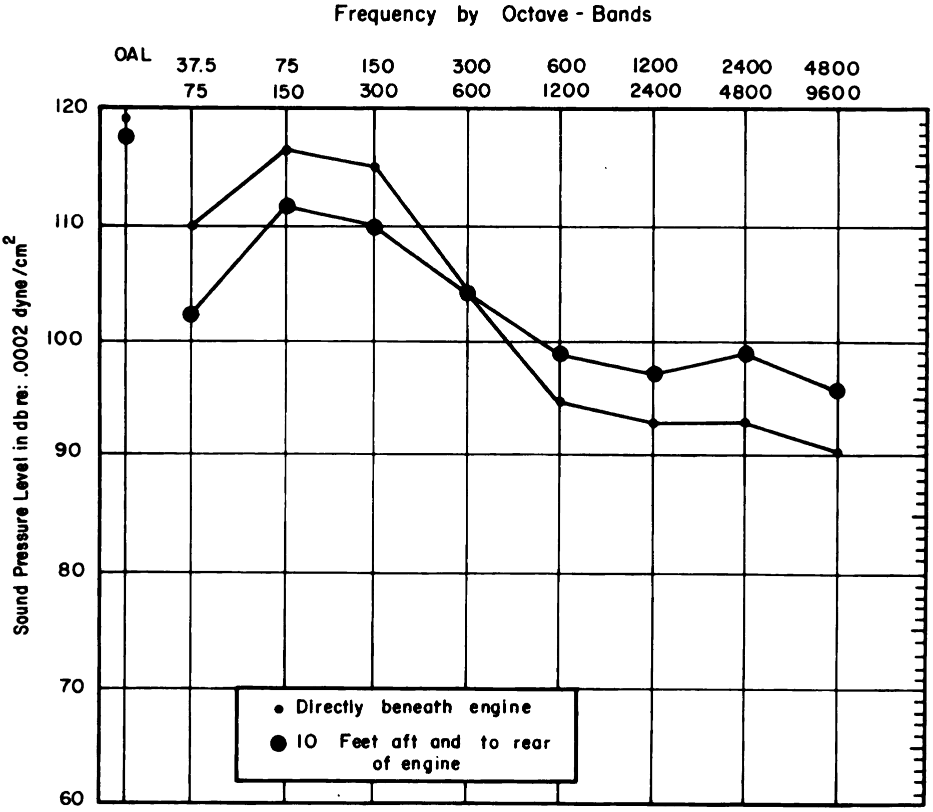

FIGURE 7-22. *Engine and exhaust noise of a large twin-engine helicopter* (GASAWAY, 1964).

PROTECTION FROM NOISE

Where noise is a hazard, there are many ways of reducing its intensity. These are outlined below:

1. Environmental Control:
    a. Reduce Source Noise Production:
        (1) Mufflers.
        (2) Damping.
        (3) Overhaul noisy equipment, replace bearings, etc.
        (4) In new construction, plan noise reduction from the beginning. Specify maximum sound levels permissible for new equipment, buildings, and spaces.

    b. Reduce Noise Transmission:
        (1) Isolate sound sources.
        (2) Use sound shields.
        (3) Absorb the sound (turn it into heat).
    c. Revise Operational Procedures:
        (1) Education of aircrew, flight deck crew, and *supervisory personnel* as to hazards and protective measures.
        (2) Rotate personnel in noise hazard environments.
        (3) Change work schedules.
2. Personnel Protection Devices:
    a. Earplugs.
    b. Earmuffs and headsets.
    c. Protective helmets.

Environmental Control

It is obvious that noise presents a very serious problem to a large number of people in the Navy and Marine Corps aviation environment. It would be ideal to control all of the noisemakers at the source by the methods mentioned above. Indeed, most of these methods are in use, at least in part. However, it is doubtful that such measures can be effective in eliminating the noise hazard any time in the near future. The Flight Surgeon must direct his concern then to the protection of personnel from the noise hazard.

Protective Devices

There are basically two types of protective devices—those worn over the ears and those which are inserted into the ear canal.

Earplugs.—The standard plastic earplug (the V–51R) is molded to fit the ear and it comes in a variety of sizes. Wads of surgical cotton are also occasionally used as ear inserts, but this practice screens out only certain frequencies, and is, therefore, ineffective in protecting hearing.

Devices Worn Over the Ears.—These include standard headsets fitted with ear cushions, flight helmets incorporating earcups, and earmuffs.

All ear protective devices tend to be more effective in attenuating high-frequency noise than noise of low frequency. The earplug, for example, may block out only 15 to 20 db or less in the low frequencies but up to 40 db in the high frequencies. Table 7–6 illustrates the noise attenuating characteristics of various ear protection devices.

TABLE 7–6

Average Octave Band Noise Level Reduction In Ear Canal Achieved By Use of Personal Protective Equipment[1]

Type of equipment	300–600 Hz	600–1,200 Hz	1,200–2,400 Hz	2,400–4,800 Hz
	db	*db*	*db*	*db*
Headset earphone covers	−7	−13	−20	−30
Standard earplugs (V–51R)	−18	−18	−25	−29
Navy earmuff Straightaway 400–9	−25	−26	−30	−38
Earplug and earmuff together	−27	−28	−30	−40

[1] From Witwer and Cole, 1961; taken from *Amer. Indust. Hyg., Quart.*, September 1952.

In fact, at sound pressure levels in excess of 140 db, no completely adequate protection can be provided, due to the bone conduction problem. The theoretical upper limit of noise protection available from earplugs and earmuffs is the sound pressure level which would be lost if the middle ear were destroyed, thus eliminating air conduction as a source of sound for the ear. This would amount to about 40 db in most frequencies. Earplugs attenuate ambient noise better than headset earphones and, when used with the headset, operate to improve the clarity of radio signals.

O'Connell (1960) indicates that ear inserts should be used with caution under flight conditions. This report provides the following explanation:

> There is an operational limitation however, which makes it mandatory that any insert defender other than dry cotton be tried out with caution under flight conditions, and that none but cotton be worn under a close-fitting helmet. If a defender worn in the outer canal seals perfectly, there will be an airtight pocket in the canal between it and the tympanic membrane. On ascent, the air in this pocket will expand and tend to equalize with the ambient pressure by pushing the plug out a bit and escaping around it. If the wearer then tightens the seal of the defender, he will again have an airtight pocket, but it will have a negative pressure on descent. The plug will tend to be sucked in, and discomfort or true vascular damage to the soft tissue of the canal may result. The term *aerotitis externa* is used to describe this condition. Experience has shown that such pathology occurs very rarely. Yet, flight personnel should be warned to watch for it, and to remove the defenders before or during descent if any discomfort is aroused. *Under no circumstances*

should ear defenders be worn under safety helmets or full or partial pressure helmets.

Pilot Protective Helmets.—There are a number of helmets in use to provide for the requirements of aviators and aircrewmen. The particular type of helmet worn is a function of the type of aircraft flown. Attack and fighter pilots and crewmembers wear one type of helmet; patrol plane pilots, another; and helicopter pilots and crew, another. Pressure suits, of course, employ still another type of helmet.

Sound attenuation characteristics of Navy protective helmets are shown in table 7–7.

TABLE 7–7. *Sound-Attenuation Characteristics of Navy Protective Helmets*

Frequency (Hz)	Mean attenuation (db)							
	AOH-1[1]	APH-5[1]	APH-6	APH-6A	APH-7	BPH-2	SPH-3	DH-76
125	5	0	3	0	1	1	3	17
250	9	−1	0	1	2	3	4	17
500	31	1	−1	3	5	10	14	29
1,000	26	16	7	3	7	21	24	31
2,000	34	6	10	5	8	22	30	38
4,000	53	20	17	14	19	38	49	50
8,000	48	23	20	23	29	25	40	41

[1] Values from Dayton T. Brown Laboratories data, provided by personnel in Bureau of Medicine and Surgery. All other values from Naval Aerospace Medical Institute.

If the type of helmet worn by a crewmember is known, examination of table 7–7 and comparison with the sound pressure level measurements which are usually available for each aircraft type in the NATOPS manual will quickly show the Flight Surgeon whether a hearing hazard exists for a particular helmet wearer. What it cannot show are such intangible variables as helmet fit, state of earcup repair, tightness of chin strap, and fullness of facial contours.

THE NAVY HEARING CONSERVATION PROGRAM

Since the health problems created by high-intensity noise have become increasingly acute, especially aboard ship, the Bureau of Medicine and Surgery has established a Hearing Conservation Program designed to preserve human hearing by preventive measures (BUMEDINST 6260.6 series). The Bureau recommends that such a program should be mandatory when noise sound pressure levels reach 85 db in any or all of the following octave bands (Hz): 300–600, 600–1,200, 1,200–2,400, 2,400–4,800. This coincides in general with conditions that exist when it is difficult to hear a loud spoken voice at a distance of one foot.

The major elements of the Hearing Conservation Program are:

1. Audiometry.
2. Personal protection measures.
3. Education of personnel.
4. Noise measurements and analyses.
5. Engineering control methods.

To carry out his responsibility for preserving the auditory acuity of aviation personnel, the Flight Surgeon must become well acquainted with the Hearing Conservation Program, and must, in fact, assure its implementation. Further information relating to the program is contained in the Instruction.

The following (from USS *Enterprise* CVA(N)–65 Instruction 6260.1) outlines the Hearing Conservation Program as it has been implemented on one carrier in accordance with the referenced BUMED Instruction.

Medical Officer Responsibilities

1. Designate hearing hazard areas onboard by definitive sound-level testing.
2. Perform audiometric evaluations upon ship's company and air group personnel working in hazard areas.
3. Make recommendations to the Commanding Officer and appropriate Heads of Departments regarding hearing conservation.
4. Educate personnel regarding the sense of hearing and its protection.
5. Issue ear plugs to personnel requiring them.

Heads of Departments Responsibilities

1. Assist the Medical Officer in meeting audiogram schedules.
2. Insure that personnel under their cognizance who work in high noise

level areas, as designated by the Medical Officer, utilize either ear plugs or sound-attenuating helmets, or both.

3. Remove personnel from high noise level working areas to lower ambient noise level working areas upon recommendation of the Medical Officer.

Air Officer Responsibilities

1. Issue sound-attenuating helmets to all personnel working in high noise level areas as designated by the Medical Officer.

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THE NOSE AND PARANASAL SINUSES

CAPT ROBERT L. KING, JR., MC, USNR

ANATOMY

The skeletal structure of the nose is composed partly of bone and partly of cartilage. The small flat nasal bones and the ascending processes of the maxillary bone give shape to the rigid upper part of the nose. The lower movable part is composed of two pairs of hyaline cartilage, the upper flat pair of which is continuous with the cartilage of the nasal septum. Certain small accessory cartilages help give shape to the alae laterally (figure 7–23).

Internally the cavity of the nose is divided into roughly equal passages by the nasal septum. Anteriorly the septum is cartilaginous. The ploughshare-shaped vomer bone (figure 7–24), firmly attached to the maxillary and palatine bones which form the floor of the nose, is the bony support of the septum inferiorly and posteriorly. Superiorly is situated the perpendicular plate of the ethmoid, and inferiorly the vomer bone. These structures are covered by mucous membrane. In the choanal region, the posterior septum is always in the midline. Anteriorly, however, one finds the nasal septum very commonly deviated to one side.

Laterally (figure 7–25) the nasal wall presents an irregular surface convoluted by the nasal turbinates. Inferiorly the lateral wall is formed by supe-

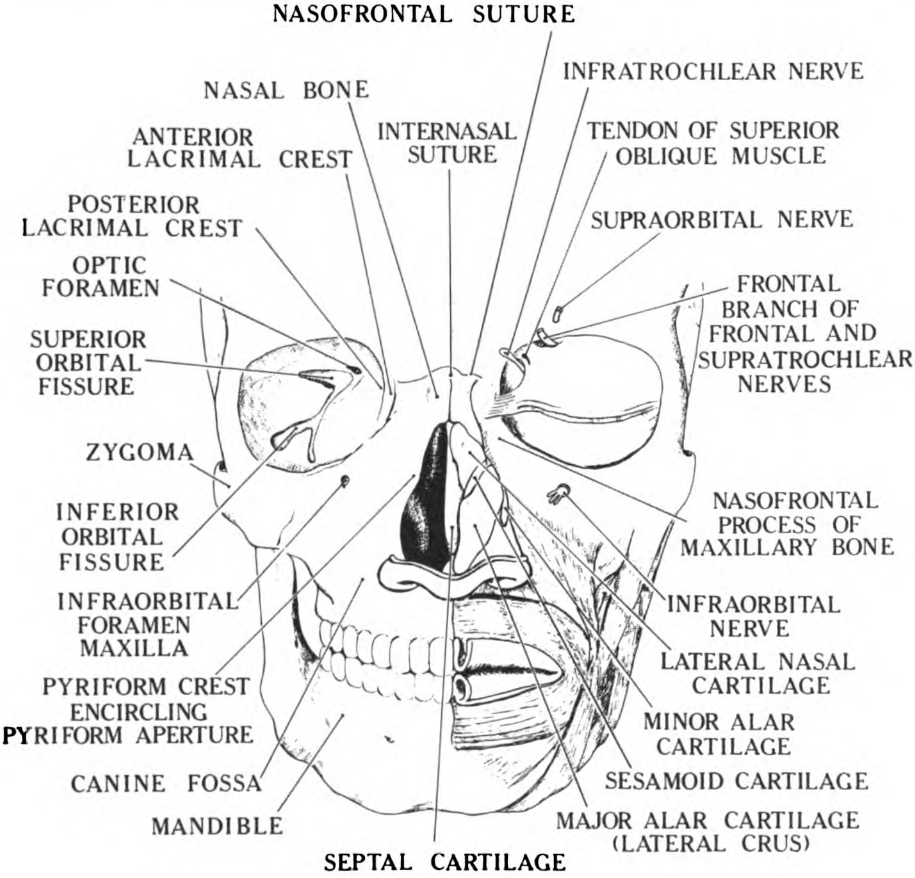


FIGURE 7–23. *Facial anatomy.*

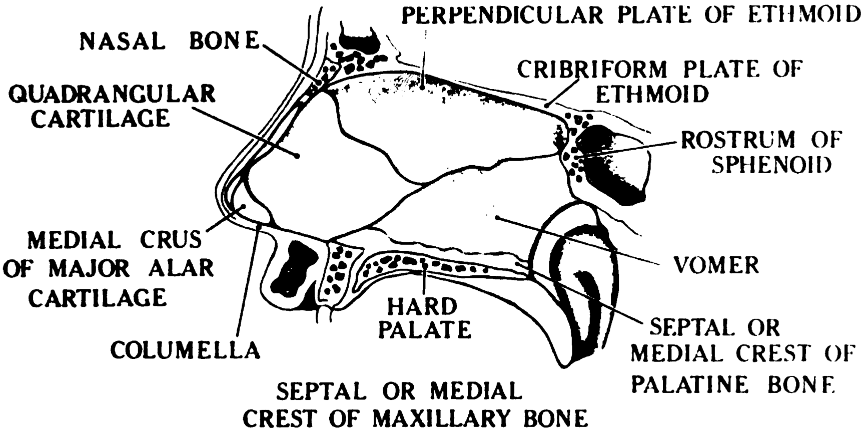

PERPENDICULAR PLATE OF ETHMOID
NASAL BONE
CRIBRIFORM PLATE OF ETHMOID
QUADRANGULAR CARTILAGE
ROSTRUM OF SPHENOID
MEDIAL CRUS OF MAJOR ALAR CARTILAGE
VOMER
HARD PALATE
SEPTAL OR MEDIAL CREST OF PALATINE BONE
COLUMELLA
SEPTAL OR MEDIAL CREST OF MAXILLARY BONE

FIGURE 7–24. *Structures of the nasal septum.*

rior maxilla. Just above this the inferior turbinate is inserted into a triangular space. Superiorly the wall is formed by the processes of the ethmoid, particularly the middle turbinate, while the lacrimal bone occupies a small space anteriorly. The posterior portion is completed by the perpendicular portion of the palatine bone and the medial plate of the pterygoid process of the sphenoid.

The lateral wall presents three or more shelflike processes which hang down into the nasal cavity, the turbinates. These are thin shells of bone covered by mucous membrane which greatly increase the mucous membrane surface area of the nasal cavity. The inferior turbinate is a separate bone and the largest of the turbinates. The middle and superior turbinates are outgrowths from the ethmoid labyrinth.

The space below the inferior turbinate is called the inferior meatus. The nasolacrimal duct drains into this space anteriorly. The middle meatus under the middle turbinate contains many important structures. The most important of these is a long narrow groove, the infundibulum. Its importance lies in the fact that into it drain all the anterior paranasal sinuses. These include the maxillary, frontal, and some of the anterior ethmoid cells. Other anterior ethmoid cells drain directly into the middle meatus unassociated with the infundibulum. A few ethmoid cells, known as the posterior ethmoids, drain into the superior meatus above the middle turbinate. The ethmoid air cells are actually divided into anterior and posterior groups by the attachment of the middle turbinate.

The roof of the nasal cavity is formed by the cribriform plate of the ethmoid capsule. It is perforated by 20 or more small openings through which pass the terminal branches of the olfactory nerve. Posteriorly and inferiorly the nasal cavity opens into the nasopharynx. Above is the curved surface of the sphenoid bone. The sphenoid sinuses drain into a narrow slit close to the nasal septum between the sphenoid and ethmoid bones called the sphenoethmoid recess. Thus the paranasal sinuses are divided into two groups, posterior and anterior. The dividing line is the attachment of the middle turbinate. Sinuses draining below the attachment of the middle

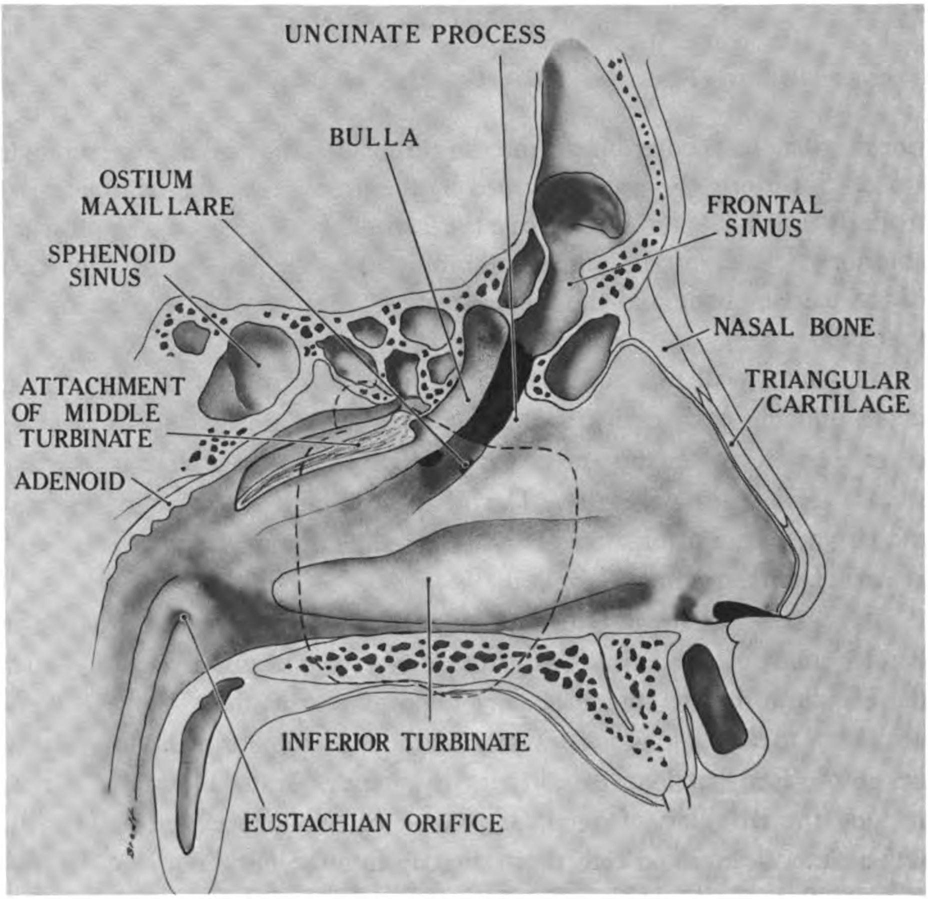


FIGURE 7–25. *The paranasal sinuses: relationship of the ethmoid cells, frontal and sphenoid sinuses and ostium of the maxillary sinus, and lateral wall of nose with middle turbinate removed.*

turbinate are the anterior group. These include the frontal, maxillary and most of the ethmoid group of cells. The posterior group includes the few ethmoid cells which empty into the superior meatus and the sphenoid sinuses which drain into the sphenoethmoid recess.

The sinuses are formed as invaginations of mucous membrane from the nasal cavity with subsequent pneumatization of the bones for which they are named. The degree of pneumatization which takes place varies greatly in different individuals. Even in the same individual the sinuses may not be of the same size on the two sides. There may be complete absence of a frontal sinus on one side with a well-developed cell on the opposite side. All sinuses must have an opening into the nasal cavity for ventilation and drainage. In about 40 percent of cases the maxillary sinus has one or more accessory openings.

The mucous membrane of the nose varies somewhat in different places. In the lower part, especially over the inferior turbinal, the mucous membrane is psuedostriated columnar ciliated with many serous and mucous glands and a rich venous blood supply. In the upper straits of the nose the mucosa becomes nonciliated and columnar in character. Cilia of the upper respiratory tract beat toward the pharynx, resulting in a continuous flow of mucous (the mucous blanket) which is deposited into the pharynx and swallowed.

Blood Supply

The blood supply to the nose, nasal fossae, and paranasal sinuses is derived from both internal and external carotid arteries. The ophthalmic artery, a branch of the internal carotid, supplies the anterior and posterior ethmoid arteries which enter the nasal fossae from the orbit by way of the cribriform area. These arteries supply the cribriform area, upper septum, ethmoid cells, and upper regions of the nasal fossae. The terminal branch of the anterior ethmoid artery is the external nasal artery which emerges on the dorsum of the nose between the nasal bone and upper lateral nasal cartilage. The external carotid artery supplies the nose and sinuses by its internal maxillary and sphenopalatine branch which enters the nasal fossae on each side through the sphenopalatine foramen, which lies just posterior to and above the posterior tip of the middle turbinate. The sphenopalatine artery divides into a medial branch, supplying the septum (nasopalatine artery) and lateral branches which ramify over the lateral walls of the nasal fossae supplying blood to the turbinates and penetrating by end branches into the sinuses. (See figure 7–26.)

Innervation

The sensory nerves to the sinuses are from the first and second division of the trigeminal nerve (see figure 7–26).

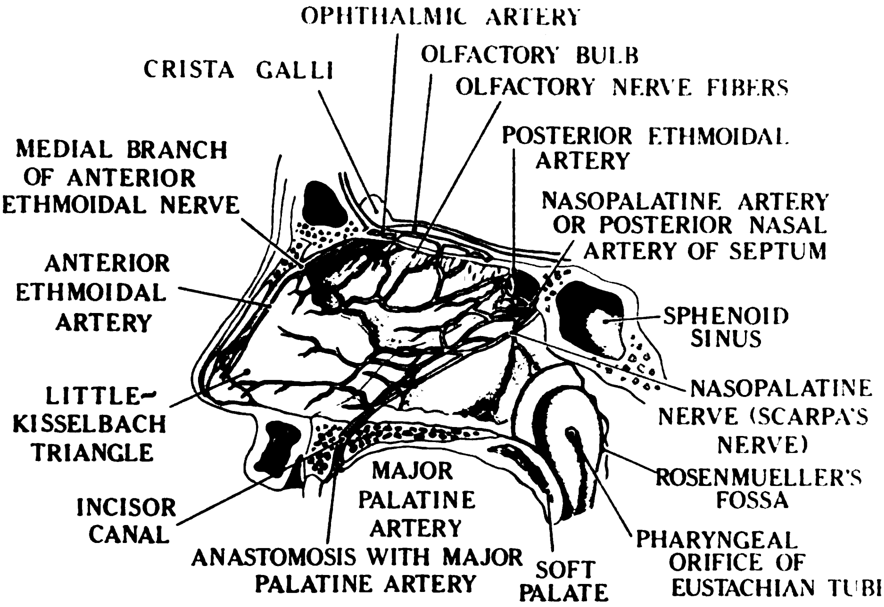

OPHTHALMIC ARTERY
CRISTA GALLI
OLFACTORY BULB
OLFACTORY NERVE FIBERS
MEDIAL BRANCH OF ANTERIOR ETHMOIDAL NERVE
POSTERIOR ETHMOIDAL ARTERY
NASOPALATINE ARTERY OR POSTERIOR NASAL ARTERY OF SEPTUM
ANTERIOR ETHMOIDAL ARTERY
SPHENOID SINUS
LITTLE-KISSELBACH TRIANGLE
NASOPALATINE NERVE (SCARPA'S NERVE)
ROSENMUELLER'S FOSSA
INCISOR CANAL
MAJOR PALATINE ARTERY
PHARYNGEAL ORIFICE OF EUSTACHIAN TUBE
ANASTOMOSIS WITH MAJOR PALATINE ARTERY
SOFT PALATE

FIGURE 7–26. *Intranasal blood and nerve supply.*

NASAL PHYSIOLOGY

Filtration, warming and humidification of the air, and preparing it for the lower air passages, are the primary functions of the nose. Olfaction is the next most important. The nasal cavity also acts as a resonating chamber in phonation. The nasal secretions possess a strong bacteriostatic property. The nose is also the origin and recipient of numerous reflex areas.

Filtration is accomplished in two ways. The vibrissae in the vestibules strain out coarse particles of dust or lint. In addition, the entire mucous membrane surface of the nasal cavity is covered by a sheet of mucous which entraps foreign matter, including bacteria. By the action of the cilia this mucous sheet is continuously being moved backward toward the nasopharynx where it is either swallowed or expectorated. The cilia move the secretions at the rate of about 5mm per minute. It is constantly being replaced by the mucous glands of the mucous membrane.

Inspired air is warmed by heat transfer from the mucous membrane, especially of the turbinates. It picks water vapor from the same source. If the mucous sheath were not being continuously replaced, the membranes would become dry, the cilia would be inactivated, and the principal functions of the nose destroyed.

Ciliary activity has been shown to be amazingly resistant to many deleterious factors such as heat and cold, acid and alkali, noxious fumes and irritating dusts. However, ciliated cells rapidly die in the absence of water; thus, continuously inspired dry air or 100 percent oxygen will alter normal functions of the nose quite rapidly. The nasal membrane then must secrete more and more mucous to make up for the surface dehydration and to replace the "mucous blanket" and keep it flowing. Central heating of houses in winter can therefore be a very important factor in altered nasal physiology, and it alone must play an important part in the causation of colds and of nasopharyngeal discharge (postnasal drip). Cold air has low water content. If taken into a house and heated, the relative humidity of this air may drop extremely low.

Similarly, many other factors can alter the normal functions ascribed to the nasal passages and paranasal sinuses. The infectious factor is self-evident and will be discussed in detail below. Nasal allergy is an extremely important factor of altered physiology. In allergic conditions, the nasal membrane is hyperesthetic and oversecreting as a result of abnormal response to an antigen or antigens. Tobacco smoke is definitely irritating and will incite disturbed function depending upon the individual's tolerance to it. Irritating dusts and fumes are factors. Endocrine upsets, particularly thyroid dyscrasias are manifest focally in the nose. Excessive alcohol intake causes intumescence of the turbinates. Truly, an intimate knowledge of nasal physiology is tantamount to sensible treatment toward all nasal and sinus conditions. Even nasal medications, incorrectly or injudiciously used, will in themselves produce subjective symptoms and objective findings. Phenylephrine HCl is particularly bad in causing a rebound reaction after two or three original applications to the nose. Other medications are even worse, but tetrahydrozoline HCl 0.1 percent does not produce the rebound phenomenon to any great extent.

The functions of the paranasal sinuses are not so well known. It has been suggested that they reduce the weight of the head, that they act as reservoirs of warmed and humidified air, and that they act as resonators for the voice. Our primary interest in these cavities is to be sure that they are adequately ventilated and drained.

DISEASES OF THE NOSE AND PARANASAL SINUSES

The Common Cold

In the approach to treatment of upper respiratory abnormalities, one must ascertain the etiology first. A common cold can be treated symptomatically only. The cold will last seven to ten days in spite of any treatment. However, if symptoms referable to the upper respiratory tract persist after this length of time, one should suspect some complication and begin the search for the site of it.

Most patients know the symptomatology of the common cold; deviations of the pattern are quickly discernible by most of us. This pattern is the well-known "dry nose, 24 hours; wet, runny, stuffy nose, three to four days; yellow discharging nose, three to four days," followed by resolution. The constitutional symptoms of malaise and fever usually occur during the wet, runny stage.

The Flight Surgeon should suspect that something is wrong when symptoms persist after the cold subsides. Persistent nasal stuffiness, unilateral or bilateral, purulent discharge, which is blown out on a handkerchief or expectorated ("P.N.D.") almost always indicate complicating sinus infection. Heavy nasopharyngeal discharge frequently causes productive cough, especially during the night when the patient is supine. In addition, affectations of the upper respiratory tract always concomitantly affect the lower respiratory tract to some degree.

The common cold is now thought to be caused primarily by a filtrable virus which is passed from person to person both by direct and indirect contact. After invasion of the virus, bacteria normally present on the upper respiratory tract become pathogenic. Although there are many predisposing factors in the etiology of the common cold, the epidemiologic aspect is the most important one. Secondary and less important factors can be chilling, fatigue, damp clothing, poor ventilation, central heating (dry air continuously inspired), and immunologic factors. Some individuals acquire very little immunity by any cold and are predisposed to another one in a short time.

The pathology involved in the common cold usually affects the mucous membrane of the entire upper and lower respiratory tract, with most of the subjective symptoms in the nasal passages. As the cold subsides, all these membranes return to normal. Complications may occur, however, in the paranasal sinuses, the Eustachian tube, and the middle ear, the larynx, trachea, and bronchi.

In general, pilots should not fly when they have a head cold. This is not meant to be a hard-and-fast rule, and the decision must be made at the time by the Flight Surgeon depending upon his opinion of the patient. However, the Flight Surgeon should constantly warn aviation personnel of the dangers of flying with "a little nose cold." The common cold is the direct cause of aerotitis media and of aerosinusitis in a great percentage of cases. It requires only a slight amount of congestion of the mucous membrane of the nasopharynx to occlude the pharyngeal opening of the Eustachian tube, or the paranasal sinus ostia.

The pilot of a jet fighter, attack, or reconnaissance aircraft should certainly be forbidden to fly with a stuffy nose of any severity. The pilot of an attack bomber (such as the A-1 series) should also be grounded. The helicopter or ASW aircraft pilot who will not exceed 500 to 1,000 feet altitude on a flight is in a different category. The pressure changes in the middle ear and in the sinuses are just not great enough to cause much trouble from aerotitis media or aerosinusitis. However, in chapter 8, it is noted that pressure *vertigo* could be produced by rapid changes in altitude due to blockage of the middle ear. Also, the indefinable factors of level of awareness and of performance unquestionably are adversely affected by having a cold. Therefore, even if pain or illness does not result from the flight, any part of the flight will be more difficult because of the cold.

Allergies of the Respiratory System

The part that allergies play in symptomatology of the respiratory system is extremely important. This subject is a vast and complicated one and can only be briefly summarized here. Suffice it to say that the Flight Surgeon should be constantly on the alert for the detection of allergic conditions in his patients. Fifty percent (or more) of the patients visiting the otolaryngologist have had, or do have, allergic rhinitis and sinusitis as the basis of their symptoms. Many "colds" are in reality an exacerbation of an allergic rhinitis. Without discussing at all the immunologic aspects of allergy, it can be said that the nasal and sinus membranes can be hypersensitive to many, many items. The symptoms produced may be not unlike those of the common cold or infectious sinusitis, making the problem that much more difficult. In spite of the fact that some attempt is made to screen out those individuals with severe allergies before they enter flight training, many pilots nevertheless turn up quite often·with allergic exacerbations at various times in various localities, and since nasal allergy is a chronic disease which is most active during the young adult years, the Flight Surgeon and the otolaryngologist are going to be confronted with this problem very often.

The outstanding symptoms of allergic rhinosinusitis are sneezing, itching, discharge, and nasal obstruction.

The pathologic physiology is mainly that of edema of the entire respiratory membrane, particularly the nasal turbinates, due to increased capillary permeability and transudation of fluid into the cells and extracellular spaces. The tissues are described as "intumescent" or "waterlogged." The respiratory membrane itself suffers an absolute increase in goblet cell formation and loss of ciliated cells. An end result can be the formation of mucous polyps; these form most often in the middle meatus and about the ostia of the maxillary sinus but seem never to form in the ethmoid air cells.

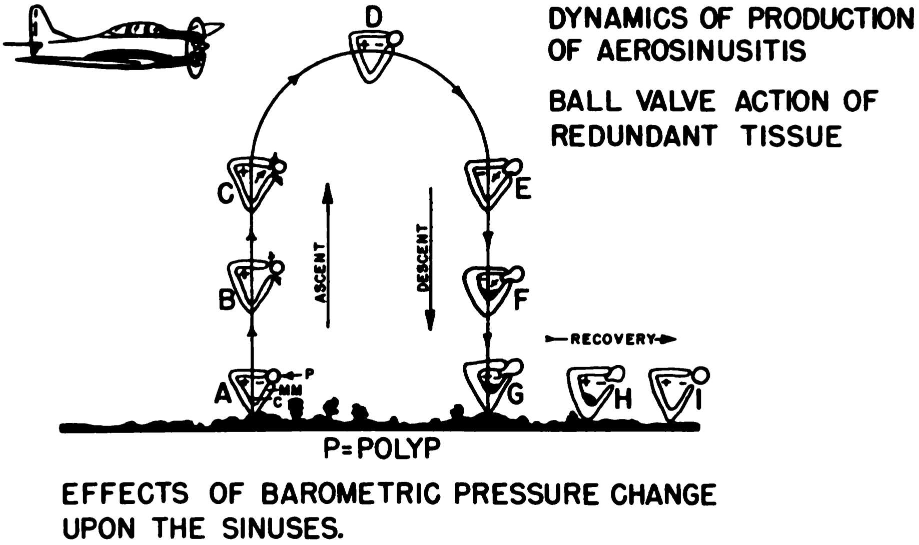
In brief, the patient is usually reacting to pollens, epidermals (dusts, linters, molds, danders, etc.), or foods. Man is potentially able to react allergically to many bacterial species. Fortunately, the development of immunity usually predominates, and invading micro-organisms are destroyed before harmful sensitivity appears. When complete immunity is not established, bacteria may exist in asymptomatic foci or chronic inflammation for long periods of time. All bacteria are capable of releasing substances which may produce allergic sensitivity in susceptible individuals. Unfortunately the presence (or absence) of bacterial allergy is impossible to prove in many clinical situations, and even the best conclusions are dependent on circumstantial evidence. This problem particularly pertains to those diseases traditionally considered allergic, such as asthma, allergic rhinitis, urticaria, and urticaria atopic dermatitis.

In the diagnosis, a careful history is paramount; in fact, an allergic history must of necessity be very detailed. Skin tests are important in the detection of respiratory allergies although their use is not often available to the Flight Surgeon. Smears of the nasal secretions are adjuvants in the diagnosis of nasal allergy versus infection. Using Hansel's stain, neutrophils and eosinophils are very nicely outlined in any nasal smear for detection under the microscope by the Flight Surgeon.

It must be remembered that asthma is often merely a manifestation of allergy in the lower respiratory tract. In the adult, asthma may be divided into two groups. One group is characterized by predictable seasonable variations, and demonstration of the specific offending allergen is usually not difficult. This type of asthma is called variably "extrinsic" or "noninfective" and responds quite well, in general, to specific allergic management. Unfortunately, a large number of patients fall into another group, "intrinsic," in which the specific etiology is not similarly demonstrable and in which skin tests are of less diagnostic value. Such patients tend to have more constant asthmatic symptoms, more often worse in winter. Frequently, exacerbations occur during otorespiratory infections. This group, in which the onset of asthma may occur after the age of 30 years, also has a strong personal and family history of allergic disease. Sinusitis and nasal polyps are common, and it is significant that the patients tend to have more eosinophils in their secretions, and a higher blood eosinophil count, than do persons with the more obvious allergic external agents. Persons with intrinsic "bronchial asthma" frequently suffer from infections of the paranasal sinuses, and active controversy wages as to whether these foci of infection are secondary to the asthmatic processes or are the primary agents which, by an allergic mechanism, can produce chronic bronchial asthma.

Aerosinusitis

Any obstruction to drainage of the sinuses results in absorption of the oxygen in the sinus cavities, stagnation of the secretions in the sinus, followed by bacterial growth and the formation of pus. These conditions are associated with headache, tenderness over the sinus involved, and usually a purulent nasal discharge. Like aerotitis, aerosinusitis usually develops during descent from high altitudes. Congestion in the nose prevents air from entering the sinus to equalize the increasing pressure of the atmosphere. Negative pressure increases in the cavity (figure 7–27). The subject is aware of discomfort


D
DYNAMICS OF PRODUCTION OF AEROSINUSITIS
BALL VALVE ACTION OF REDUNDANT TISSUE
C
E
ASCENT
DESCENT
B
F
RECOVERY
A
P
MM
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EFFECTS OF BAROMETRIC PRESSURE CHANGE UPON THE SINUSES.

FIGURE 7–27. *Mechanics of aerosinusitis.*

followed by severe pain over the sinuses involved, usually above or below the eye. The accompanying pain may be so severe as to pose a hazard to landing.

Jet aircraft have high descent rates which tend to exacerbate the problem. They also have high fuel consumption at low altitude, so that the time available for attempts at therapy in flight is not very long, if indeed the pain is so severe as to cause the pilot to be concerned about attempting a landing. The steady breathing of very dry 100 percent oxygen also exacerbates the problem by altering nasal physiology and, of course, this effect is most noticeable at the end of the flight, when aerosinusitis is most likely to occur.

Generally speaking, the Flight Surgeon is likely to find out about a case of aerosinusitis only after the landing. If, however, the pilot has such severe pain that he considers it unwise to attempt a landing (especially aboard ship), he will probably be directed to return to a higher altitude to attempt to find relief. If the pain still does not disappear, he may be ordered to divert the flight to a "Bingo" field. The Flight Surgeon's role during such a situation is, of course, only that of an adviser and consultant.

Many Flight Surgeons recommend that aviators carry an approved inhaler containing a mild nasal vasoconstrictor in their flight clothing during all flights, specifically for use in such emergencies. Use of such self-medications during flight can usually produce within a short time sufficient decrease in nasal and Eustachian swelling and edema that pressure can be equalized and a successful landing performed. All such cases should be reported to the Flight Surgeon for examination and treatment immediately after landing. Treatment includes shrinkage of the nasal membranes and aspiration of secretions. The best treatment is prevention—grounding aviators who have colds or nasal stuffiness of whatever etiology.

Incidence of aerosinusitis in low pressure chamber subjects was 1.5 percent in a series of 2,351 individuals given LPC indoctrination (Adler, 1964). Location of the pain is almost entirely frontal or maxillary, or both, with 70 percent frontal, 10 percent frontal and antral, and 19 percent antral alone.

The prognosis for aerosinusitis is good. Almost all cases are returned to full flying duties as soon as the acute nasal condition is resolved.

Treatment of diseases of the nose and sinuses has as its objective the return of adequate ventilation and drainage. The common cold is treated symptomatically with vasoconstrictors and salicylates. If the condition appears allergic in etiology, the diagnosis can be substantiated by means of a nasal smear, and appropriate antihistamines administered.

Sustained-release medications, such as Triaminic tablets and Ornade spansules are excellent in such situations, but the aviator should be grounded and closely supervised while taking them. In suppurative conditions of the nose and paranasal sinuses that are accompanied by constitutional signs (fever, leucocytosis, etc.), antiobiotics might be prescribed.

Deflection Of The Nasal Septum

Inflammatory conditions of the nose and sinuses can be aggravated by abnormalities of the nasal septum. The septum is rarely absolutely straight; in reality, a deviated nasal septum is a "normal abnormality" of the Caucasian race. Such deflections are not important unless they interfere with the normal functions of the nose. Spurs or ridges in the lower portion, opposite the inferior meatus, may cause no inconvenience unless they are located far anteriorly in the region of the limina vestibularis. Abnormalities higher up, pressing on the middle turbinate, may well obstruct the drainage of the anterior group of sinuses which empty into the middle meatus. Contact of any portion of the nasal septum with the lateral nasal wall in the region of the middle meatus or middle turbinate almost always indicates the necessity for a submucous resection.

Trauma to the nose at any time of life is not uncommonly followed by injury to the anterior septum in the columellar region. This quadrangular cartilage may be twisted or fractured, displacing it from its groove in the midline of the columella. In any severe trauma to the nasal pyramid, which results in fracture of its bones, the nasal septum should be carefully examined for concomitant injury.

Epistaxis

Most nosebleeds occur from the area on the septum just inside the vestibule, known as Kiesselbach's area. Here the different arterial sources to the septum anastomose in a very vascular plexus. Trauma is perhaps the most common direct cause, but various diseases of the nasal membrane, violent blowing of the nose, sneezing, the inhalation of irritating dust, and tumors all contribute. Epistaxis from the various types of cardiovascular disease are not frequently seen among flying personnel.

Severe bleeding from the nose can also occur from the ethmoid arteries as they enter the superior meatus in the cribriform area, or from the sphenopalatine artery which enters the nose far posteriorly.

The bleeding point may then be located and controlled by a small pack of cotton soaked in topical adrenalin. Before attempting to control severe bleeding from the nose, the physician should have all the necessary equipment set up before him. This should include head mirror, nasal speculum, bayonet forceps, aspiration machine and nasal suction tip, vaseline gauze, cotton, and adrenalin. Never cauterize an actively bleeding vessel. Stop the bleeding first with packs and adrenalin, even if it is necessary for the pack to remain in place for 24 hours. Then the ulcerated area may be judiciously cauterized with 5 percent chromic acid, 50 percent silver nitrate, or 50 percent trichloroacetic acid. Chemical cautery to the nasal membranes should never be applied on "soppy" sponges. Actual (electric) cautery should not be used except in the hands of the experienced. Severe, uncontrolled posterior nasal hemorrhage may require a postnasal pack, but the latter is extremely uncomfortable to the patient and should be relied upon only as a last resort. Often the control of a severe nasal hemorrhage will try the patience, skill, and ingenuity of the surgeon. Ideally, the best technique in the control of epistaxis would be to use what might be called "spot" packing of only the bleeding area under direct vision. This can be done when the bleeding is in the anterior nares, such as that which originates in Kiesselbach's plexus (Little's area). Unfortunately, the bleeding which occurs in the posterior nares is usually either from the sphenopalatine artery, which is a distal end-artery of the internal maxillary, or from the anterior or posterior ethmoid arteries, which are well-nigh impossible to visualize directly. In the massive epistaxis which occurs from these two areas, the only technique is to put in a firm-fitting postnasal pack and then exert heavy anterior packing pressure, filling both nares with the anterior vaseline gauze packed firmly into the nose. A useful medical adjunct of fairly recent origin in the treatment of epistaxis is that of Premarin, injected intravenously slowly, 20 milligrams initially, and repeated at six-hour intervals.

Infectious Sinusitis

Infectious sinusitis may be present at all ages and respects neither season nor climate; the majority of sinus infections follow an acute upper respiratory infection, particularly the common cold. Other infections may be caused by infected upper molar teeth (the antrum) or by swimming and diving (the frontal sinus). The extent and persistence of the condition depend on two major physiological principles, ventilation and drainage, and treatment is always directed toward the improvement of aeration and drainage. Antibiotics and sulfonamides are helpful as adjuncts in the treatment of some cases of infectious sinusitis, but adequate drainage through natural or surgical openings must be established before one can expect a cure.

Maxillary Sinusitis.—In acute maxillary sinusitis the predominant symptom is severe neuralgic pain referred to the cheek on the affected side, above the eye, or in the upper dental arch. There is tenderness to pressure over the involved sinus. As the condition becomes purulent, the symptoms are aggravated. If the disease is secondary to a dental infection, a foul odorous discharge is characteristic. Headaches are common, particularly when the natural ostium is occluded. Nausea, vomiting, and dizziness may occur. There is usually discharge oozing from under the middle turbinate.

Chronic maxillary sinusitis results when the acute condition fails to clear up under local treatment. The exudate becomes organized, the mucous membrane lining thickened and redundant. When in doubt, irrigation of a suspicious appearing antrum aids in diagnosis. X-rays also are a necessity in doubtful cases.

In establishing ventilation and drainage, shrinkage of the nasal membranes, followed by mild suction, is helpful in many cases. Steam inhalations, infrared, and diathermy all have their place. Once the acute condition has subsided, irrigation of the antrum, either through the natural ostium or by means of a trocar under the lower turbinate, should be done periodically (figure 7–28). If irrigation twice a week fails to show signs of improvement after a few weeks, more radical measures become necessary. If the infection is dental in origin, it is useless to attempt to cure the sinusitis while the offending tooth remains in place.

Frontal Sinus Disease.—Symptoms of frontal sinus disease are frontal pain and tenderness, sometimes referred to the temporal regions, and headache, which is frequently known as "union headache" because of its origination sometime in the midmorning and its clearing late in the afternoon. The pain is aggravated by blowing the nose or coughing. Fever, leucocytosis, and generalized toxicity may be present. If the nasofrontal duct is obstructed, fistulous tracts may develop. The bony floor of the sinus is quite thin and it is here that fistulas most commonly occur. But the pus may break into the ethmoid labyrinth, the nasal cavity, the orbit, or the cranial cavity, resulting in meningitis and brain abscess. Osteomyelitis of the skull is one of the most feared and dangerous complications.

If the condition fails to respond to local treatment along with systemic antibiotics, surgical measures must be considered. Infraction of the middle turbinate should be done to improve drainage into the middle meatus before any other surgical procedure is attempted. If, with such simple procedure

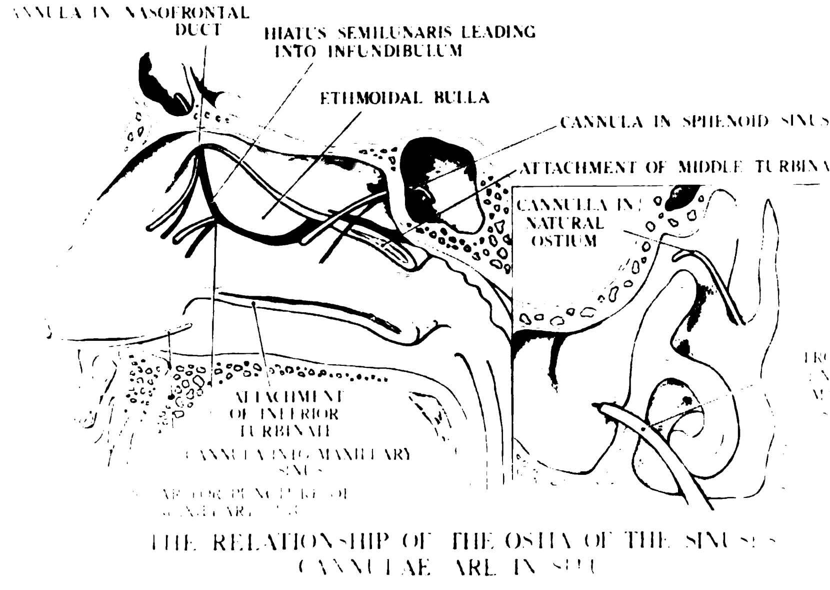
CANNULA IN NASOFRONTAL DUCT
HIATUS SEMILUNARIS LEADING INTO INFUNDIBULUM
ETHMOIDAL BULLA
CANNULA IN SPHENOID SINUS
ATTACHMENT OF MIDDLE TURBINATE
CANNULLA IN NATURAL OSTIUM
ATTACHMENT OF INFERIOR TURBINATE
CANNULA INTO MAXILLARY SINUS
THE RELATIONSHIP OF THE OSTIA OF THE SINUSES
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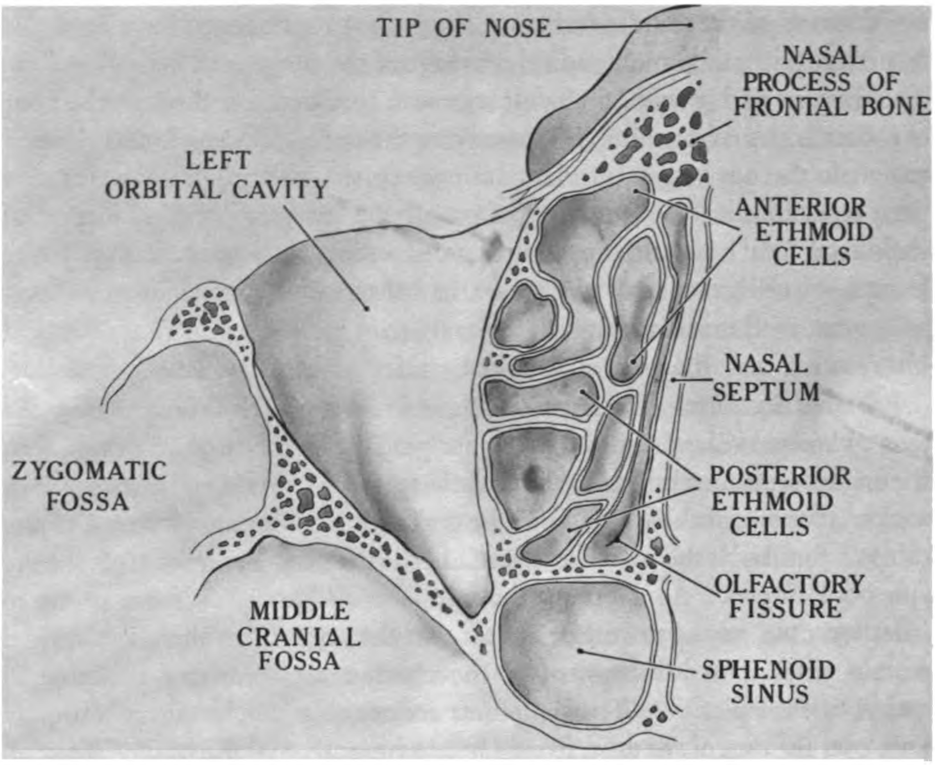
Figure 7–28. *Cannulization of paranasal sinuses.*

as infraction of the middle turbinate, the patient continues to have an active frontal sinusitis, then more radical drainage of the sinus must be undertaken. The simplest and perhaps the safest approach is to trephine through the floor of the sinus. A rubber drain is sutured into the trephine opening and allowed to remain there as long as purulent drainage persists or until drainage into the nose is established. Fortunately, most of the cases respond to intranasal shrinkage, local heat, and the systemic use of antibiotics. In acute suppurative frontal sinusitis, one may not persist in conservative management as long as one can with maxillary sinusitis. Severe pain indicates imperative surgical intervention and the patient should be referred to a specialist if possible.

Ethmoid Sinusitis.—The anterior ethmoid cells are in close proximity to the frontal and maxillary sinuses (see figure 7–29). Furthermore, all drain into the hiatus semilunaris, under the middle turbinate. As a consequence it is seldom that one of these sinuses is diseased without some involvement of the others. Similarly, the posterior ethmoid cells may become infected from the sphenoid sinuses. As a group, the ethmoids are probably more prone to infection than any of the other sinuses. In the acute form there is always a certain amount of involvement of the mucous membrane of the nose to complicate the picture. The symptoms are not clear cut, but there is usually pain over the root of the nose, frontal headache, and nasal discharge. Transillumination and external pressure over the frontal and maxillary sinuses help to exclude them as the source of the discharge. A swollen upper lid is associated with frontal sinus disease, while swelling of the lower lid is characteristic of ethmoiditis. Swelling of both lids indicates combined involvement. Proptosis of the eyeball may result from rupture of an ethmoid infection into the orbit. Movement of the eyes may be painful due to a periostitis about the pulley of the superior oblique muscle. The appearance of the middle turbinate is frequently helpful in diagnosis of ethmoid disease. In the early stages of the disease the middle turbinate is red and swollen. Later it may assume a polypoid appearance with markedly thickened mucous membrane. Pain is rare in the chronic form but there is a sensation of chronic dryness and secondary pharyngitis which causes discomfort.

In treatment, conservative measures are tried first: shrinkage, suction, chemotherapy, and local heat. If indicated, a submucous resection or infracture of the middle turbinate should be done to improve drainage from the middle meatus. Concomitant infection of the antrum must be attended to by frequent lavages. Surgical manipulation of the ethmoid labyrinth is infrequently necessary today. However, an intranasal ethmoidectomy is



RELATIONSHIP OF THE ETHMOID LABYRINTH AND SPHENOID SINUS VIEWED FROM JUST BELOW THE BONY ROOF OF THE NOSE.

FIGURE 7–29. *Ethmosphenoid sinus relationship.*

indicated when there is extensive polypoid degeneration of the lining membranes. Radical external ethmoidectomy is reserved for removal of malignant tumors.

Sphenoid Sinus Disease.—Inflammation of the sphenoid sinuses is uncommon but may be a direct extension from disease of neighboring sinuses, the nasal mucous membrane, or from the nasopharynx. The ostia of the sphenoids open into the sphenoethmoid recess and may become obstructed by swelling of the mucous membrane. The symptoms are variable but may consist of a deep-boring headache, occipital or parietal, inability to concentrate, associated with malaise and anorexia. In the chronic cases, symptoms are so variable that certain writers have even described a characteristic pain in the arch of the foot. Symptoms may appear as ocular, aural, intracranial, mental, or systemic manifestations. Pain is frequently referred to the mastoid area. Rupture may take place into the orbit, the ethmoids, or the cranium with the development of a rapidly fatal meningitis. Cavernous sinus thrombosis is another feared complication.

Examination of the nose and nasopharynx may show little or nothing. In chronic cases there is usually hypertrophy of the lateral lymphoid bands in the pharynx with the presence of thick, greenish, tenacious exudate. Postrhinoscopy may show the pus oozing downward from the ethmosphenoid recess on one or both sides. If orbital extension has occured, there may be proptosis, paralysis of the extraocular muscles with chemosis and swelling of both lids. Examination of the nose may show pus coming from above the middle turbinate. The X-ray is imperative in the diagnosis.

Treatment of sphenoid disease follows the pattern laid down for the other sinuses. Surgical intervention must be done with extreme care because of the close proximity of the sphenoid sinus to the orbit, the optic nerve and chiasm, the cavernous sinus, the meninges, the pituitary body and the internal carotid artery. As dehiscences may occur in the spenoid bone, any of the above may lie under the mucous membrane of the spenoid sinus.

THE PHARYNX AND LARYNX

CAPT Robert L. King, Jr., MC, USNR

Infections of the Mouth and Pharynx

The pharynx is part of both the alimentary tract and the respiratory tract and is subject to lesions peculiar to both of these systems. The functions of the mouth and pharynx include mastication, salivation, and formation of speech, and it is an important auxiliary respiratory system. Any lesion interfering with these natural functions is disqualifying. We can mention but a few of the more common conditions which not only cause discomfort to the patient but also are an important cause of lost man-hours.

Acute Pharyngitis

As the pharynx is directly continuous with the nose, it is to be expected that a certain degree of pharyngitis will accompany any infection of the upper respiratory tract. It is a sufficient cause for grounding flying personnel. In addition to the local treatment to the nasal membranes, hot saline irrigations of the pharynx are comforting and beneficial. Analgesic drugs (codeine and aspirin), forced fluids, and specific chemotherapy should be used when susceptible organisms are found. The most common cause of sore throat in tonsillectomized adults is apt to be an acute epipharyngitis secondary to chronic sinus disease, particularly chronic ethmoiditis.

Tonsillitis and Sore Throat

Acute follicular or lacunar tonsillitis is frequently seen among military personnel, particularly during the winter months of the temperate climates. Tonsillitis and "sore throat" are generally used synonymously by the patient, yet they are different entities as far as the physician is concerned. The treatment of acute follicular tonsillitis is still a problem in spite of antibiotics and chemotheraphy now available. This is apparently due to antibiotic- and chemotherapeutic-resistant organisms. A physician treating acute tonsillitis, therefore, should treat the patient as if he had no antibiotic or chemotherapeutic agents: by means of pharyngeal irrigations, bed rest, and fluids. By this is meant, the patient should be treated actively in spite of the dependency of antibiotic aid.

If neglected, follicular infection of the tonsils can perforate the tonsillar capsule and peritonsillar abscess or "quinsy" results. Quinsy cases should be treated vigorously with high dosages of pencillin, at least 600,000 units three times daily, hot saline throat irrigations, adequate relief of the severe pain, bed rest, and liquid diet. If a peritonsillar abscess shows evidence of "pointing," incision and drainage under local anesthesia is definitely indicated and generally gives the patient early relief of his pain and trismus. Local anesthesia is obtained best by means of a topical application of the anesthetic in the region of the sphenopalatine ganglion at the posterior end of the middle meatus.

Tonsillectomy

Tonsillectomy is indicated when it is evident to the physician and the patient that the latter would be better off without the tonsils. In other words, the Flight Surgeon should pay attention not only to the appearance of the tonsils, but also to the complaints and history of the patient. How much trouble the infected tonsils cause the patient is the important point. The question of whether or not tonsillectomy is indicated is constantly being met. It is well for the Flight Surgeon to have a routine for the handling of these cases. A history of repeated attacks of tonsillitis or peritonsillar abscess, hypertrophied tonsils which obstruct breathing or deglutition, or tonsils suspected of being foci of infection are all adequate indications for tonsillectomy. Tonsillectomy should not be performed immediately after an acute infectious process but delayed for at least six weeks following complete healing of the infectious process.

Chronic sore throat is usually due to irritation rather than infection of the mucous membranes, although a low-grade chronic ethmoiditis or maxillary sinusitis may give rise to recurring sore throats. An outstanding example of irritant pharyngitis is that due to smoking. Overuse of tobacco is a very common cause of sore throat. Elimination of the underlying cause will frequently result in relief of recurring sore throat.

THE LARYNX

Anatomy

The larynx consists of a number of cartilages held together in a boxlike tubular arrangement of muscles, membranes, and ligaments. The whole lies in the middle of the neck below the hyoid bone in front of the lower half of the cervical spine. The larynx is bound in front and at the midline directly by skin and the cervical fascia; laterally, by the thyroid gland, the sternothyroid, thyrohyoid, omohyoid and inferior constrictor pharyngeal muscles; behind by the hypopharynx. Above, it opens into the aditus laryngis into the pharynx, and below it continues into the trachea. The larynx is larger and the thyroid cartilage assumes a more pointed shape in the male than in the female.

Physiology

The larynx has two principle functions, biological and social. Its biological function, first from a phylogenetic standpoint, relates to its valvular character and consists of respiratory control and protection of the airway. Its *social* or "expressive" function is phonation, or the production of voice, and may be either voluntary or involuntary, according to the action of the vocal mechanism. Acting as a valve, the larynx is the protective mechanism which prevents food, foreign materials, and secretions from entering the lower respiratory tree. Other valvular functions contribute to the delicate control of intrathoracic pressure, thereby assisting in the regulation of carbon dioxide elimination, the degree of valvular distention, respiratory reflexes, and of the blood flow through the chest, especially in the venous return. In aviation, the proper fixation of the thorax in order to do a Valsalva maneuver is dependent upon the accomplishment of the closure of the larynx. An intact larynx is therefore an essential in aviation.

Phonation.—Sounds in the human are produced first by the initiation of a more or less steadily moving column of expired air. Second, there is a complicated interaction of this column of air in the vocal chords which results in the production of a progressive vibratory airway. These two phonation actions may occur together, as in the production of all voiced sounds, or the phonatory action may take place by the use of the breathstream alone without vibratory movement of the vocal chords, as in the production of the voiceless sounds like the aspirated H and the fricative consonants, S, SH, F, and TH as in THIN. The production of voiced sounds might be likened to the blowing of a column of air across a pop bottle; where the column of air blowing across is broken up and turbulence results, producing sound. This is the basic mechanism of phonation rather than the vibratory mechanism as in the production of a tone on a violin string.

Innervation.—All muscles of phonation and respiration of the larynx are supplied by the inferior laryngeal branch of the recurrent laryngeal nerve (vagus and bulbar accessory complex), except the cricothyroid (anterior tensor) which is supplied by the external (motor) branch of the superior laryngeal nerve. The sensory innervation of the larynx is supplied by the internal branch of the superior laryngeal nerve.

Blood Supply.—The superior laryngeal artery, a branch of the superior thyroid, accompanies the internal laryngeal nerve; the inferior laryngeal artery, which takes its origin from the inferior thyroid, accompanies the inferior laryngeal nerve. Both of these laryngeal vessels anastomose in the laryngeal wall to supply muscles, glands, and mucosa of the larynx.

Examination Of The Larynx

The larynx should be examined routinely, even though no outstanding phonatory or respiratory difficulty is manifest. To understand the importance of laryngology, one needs only to realize that, for example, in laryngeal tuberculosis hoarseness is not necessarily present. Even malignant lesions of the epiglottis, false cords, or epiglottic folds may cause no hoarseness or dyspnea until late in the disease. The technique of mirror examination is as follows: The light from the examiner's head mirror is reflected into the mouth; the patient is asked to protrude the tongue, around the tip of which a piece of gauze is wrapped. The tongue is grasped by the examiner between the index finger above and *rolled* over the thumb of the same hand below. This maneuver is important to prevent pulling the tongue sharply down against the teeth. Gentle traction is made while the patient is told to breathe through the mouth in short, quiet gasps. A laryngeal mirror, warmed so as to prevent condensation of moisture on its surface, is held in the right hand and introduced back into the pharynx, at the same time having the patient say "Ah." This raises the palate and permits the mirror to be introduced higher and further back toward the posterior wall of the pharynx, lifting the uvula in the procedure. Special care should be exercised to avoid touching the base of the tongue with the mirror since this is a sensitive area which would initiate an immediate gag reflex; gentle, firm pressure on the posterior pharyngeal wall is not contraindicated, however, and is a preferred technique to the very light touching of the uvula and soft palate which frequently initiates the gag reflex. Indirect mirror laryngoscopy is frequently preferable to direct laryngoscopy in that a panoramic view of the entire larynx with its interrelationships may be obtained by mirror use whereas direct examination limits the field and perspective of the examiner.

Diseases Of The Larynx

Since the number of diseases of the larynx which occur only infrequently is fairly large, a laryngologist is certain, at one time or another, to observe at least one of these. Consequently, conditions less commonly encountered must be kept in mind constantly in making a differential diagnosis of laryngeal pathological processes. Most forms of laryngeal disease have symptoms in common which are due to interference with the essential functions of phonation, respiration, and deglutition.

Hoarseness.—Hoarseness is defined as roughness or discordance in the quality of the voice. The voice is influenced either by alteration of its pitch or its intensity, or both. Hoarseness as a symptom is usually grouped into two general all-inclusive classes on the basis of pathogenic factors alone.

1. Those factors which influence the voice (either intensity, pitch, or quality) without a pathologic process in the larynx itself.

2. Those locally acting causes which produce the hoarseness by a lesion within or directly external to the laryngeal chamber.

Acute Laryngitis.—Acute laryngitis is probably the most common of laryngeal diseases. It is the result of an acute local hyperemia, secondary to such things as overuse of the voice, mechanical or chemical irritation, climatic changes, or contiguity of structure. Infections may also be bloodborne. Men appear to be more susceptible to laryngeal irritations than women, doubtless because they are commonly exposed to occupational injuries and indulge to a greater degree in deleterious habits. For example, excessive use of the voice, ingestion of hot, spicy foods, varying diets, irregular modes of living, and abuse of alcohol and tobacco are frequently causes of laryngitis. Acute laryngitis may be hyperemic, exudative, or ulcerative in nature. All parts of the larynx are likely to become involved, including the subglottic area. Swelling of the subglottic area is at times sufficient to cause its projection into the borders of the chords. Slight edema of the chords frequently is present, occasionally becoming severe enough to be alarming because of the resultant respiratory obstruction. When the vocal chords become edematous, they cause pressure against one another and produce decubitus ulcers. Contact ulcers develop near the vocal process of the arytenoids as a result of excessive use of the voice during an acute stage of laryngitis, and perichondritis ulceration followed by necrosis may occur. Membranous formation is occasionally observed. Later, when the process subsides, scarring and possible deformity may result. Treatment of acute laryngitis is primarily one of prophylaxis; care of the nasal and pharyngeal conditions is to be emphasized. It is generally the professional voice users who seek the advice of the physician, in order that their work may not suffer from any interruption. They are, for this reason, poor patients to deal with at the onset, not being fully cooperative but rather flighty and with ideas of their own as to what constitutes the proper care of the voice. In general, therapy of acute laryngitis consists of what may be called absolute voice rest; absolute voice rest means absolutely no talking, no whispering, no smoking, no drinking of alcoholic beverages. Spicy foods are to be eliminated from the diet. The patient should be ordered to bed in a warm, moist, well-ventilated room to avoid irritating vapors. Foods should be neither hot nor cold, though many patients are made more comfortable by swallowing ice cream or cracked ice. Aspirin is prescribed if there is fever. Treatment of contributory diseases must not be overlooked. In severe cases, administration of antibiotics may be indicated.

Chronic Laryngitis.—Repeated attacks of acute laryngitis frequently lead to chronic pathological changes within the laryngeal structures, which are primary in origin. Such factors as faulty or improper use of the voice (singers, public speakers, hucksters, and peddlers), exposure to adverse conditions (workers in large offices) and to dust and fumes (stonecutters and chemical workers), commonly predispose the larynx to chronic irritative phenomena. The latter group of irritants are described as external factors. Chronic laryngitis may take the form of such things as small bilateral vocal nodules at the junction of the anterior and middle thirds of the vocal chord to the large sessile polyp of the vocal chord. Occasionally, if the patient has utilized his voice in spite of the warning symptoms of hoarseness, the initial infiltration of dilating blood vessels which attempt to heal the irritative reaction may rupture, causing a hemorrhagic laryngitis. These hemorrhagic swellings in the submucosal tissue may become organized. Usually such changes are visible on the anterior surface of the chords or on the edge, most often extending into the submucosa.

Any affliction of the phonotory ability of an aviator is cause for rejection of that person from an aviation program. The use of the voice for radio transmission of voice directions in aviation is imperative. Even more important is the necessity for the pilot to have an adequately functioning larynx in order to talk while utilizing positive pressure breathing.

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CHAPTER 8

The Nonauditory Labyrinth in Aerospace Medicine

FRED E. GUEDRY, JR., PH. D.

STRUCTURE OF THE NONAUDITORY LABYRINTH
FUNCTIONAL ASPECTS OF THE NONAUDITORY LABYRINTH
  Semicircular Canals
  Otoliths
EVALUATION OF PILOT DISORIENTATION
  History
  Examination
  Preventive Measures
REFERENCES

MOST PILOTS have had experiences of disorientation in flight when vision has been degraded by darkness or weather conditions (Clark & Graybiel, 1955; Clark & Graybiel, 1957; Clark & Nicholson, 1954; Page, 1950; Vinacke, 1946; Vinacke, 1947). Accidents and near-accidents have been attributed to various forms of spatial disorientation (Clark & Graybiel, 1957; Eastwood & Berry, 1960; Neely, Zeller & Normand, 1958; Nuttall, 1958; Nuttall & Sanford, 1956; Ogden, Jones, & Chappell, 1966). The vestibular system of the inner ear responds to movement more or less like an inertial guidance system, and it normally provides sensory data and reflex activity which aid in coordinated movement. However, in some unnatural maneuvers encountered in aircraft, or in the event of labyrinthine disease, this system can be the source of considerable disorientation. For this reason, it has a special significance in aerospace medicine.

STRUCTURE OF THE NONAUDITORY LABYRINTH

The vestibular system of the inner ear lies in the petrous portion of the temporal bone in each side of the skull. As shown in figure 8–1, it consists of three semicircular canals which connect to a common chamber called the utricle. The semicircular canals lie in three planes approximately at right angles to one another. The planes of the two vertical canals are 45° from the midsagittal plane of the skull. The lateral or horizontal canals lie approximately in a plane defined by lines drawn from the outer canthi of the eyes to the tragi of the ears. Within the bony canals are membranous canals which are separated from the bony canals by another fluid called perilymph. The bore of a membranous canal is between 0.1 and 0.2mm. The lengths of the canals are about 13, 15, and 12mm for the superior, posterior, and horizontal canals, respectively, in man. At one end of each canal is an enlarged chamber called the ampulla. Protruding into the ampulla and forming a fluid-

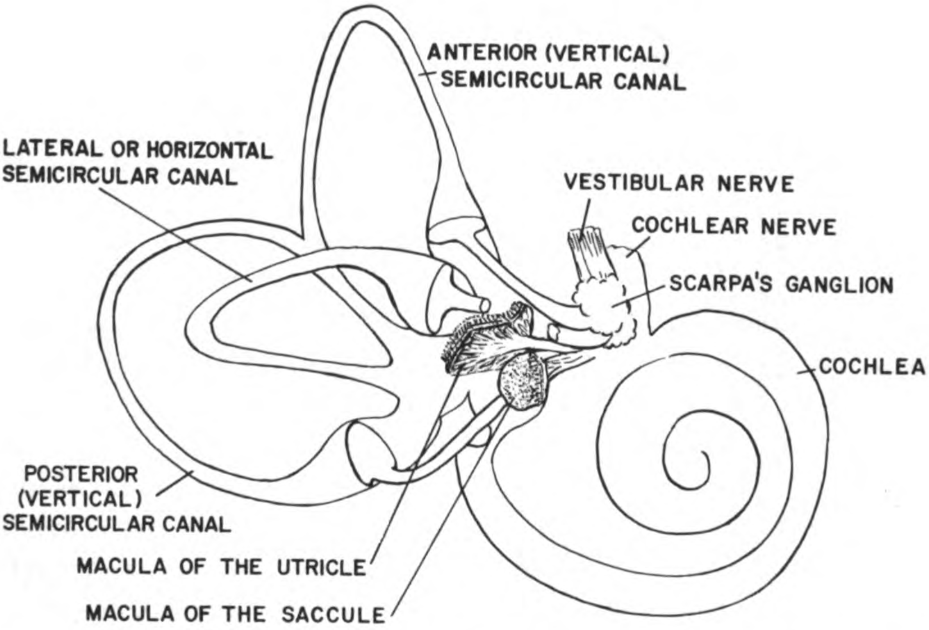
ANTERIOR (VERTICAL) SEMICIRCULAR CANAL
LATERAL OR HORIZONTAL SEMICIRCULAR CANAL
VESTIBULAR NERVE
COCHLEAR NERVE
SCARPA'S GANGLION
COCHLEA
POSTERIOR (VERTICAL) SEMICIRCULAR CANAL
MACULA OF THE UTRICLE
MACULA OF THE SACCULE

FIGURE 8–1. *Lateral view of the right inner ear. Enlarged end of each semicircular canal is the ampulla. The crista is a transverse ridge at the base of each ampulla, which is supplied by a branch of the vestibular nerve. Drawing adapted from Krieg,* Visualization of the Functional Anatomy of the Ear, *published by the Aurex Corp.*

tight hinged valve is the cupula (shown in figure 8–2 below), a gelatinous structure containing hairs which arise from a crest which traverses the base of the ampulla. This crest, the crista, contains hair cells which transduce the physical displacement of the hairs within the cupula into neural activity. The hair cells and nerve endings in this crest have been studied electrophysiologically and by electronmicroscopy, and it has been argued that some cells are influenced by feedback from the central nervous system (CNS) and perhaps from within the system of hair cells themselves (Wersäll & Flock, 1965). Therefore, this system, like other sensory sytems, is more complex than a simple detection organ, and its sensitivity may be under central nervous system control. The specific gravity of the endolymph and the gelatinous cupula are believed to be the same or very nearly the same, which means that the semicircular canals would be relatively insensitive to *linear* acceleration. Hence this system is believed to be primarily sensitive to *angular* acceleration. Recently, however, questions have been raised about this point, and it is currently under investigation (Guedry, 1966).

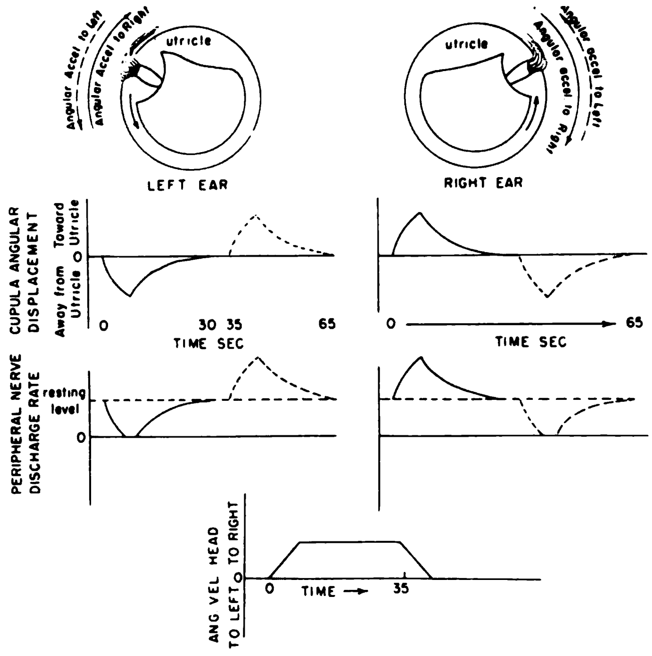

FIGURE 8–2. *Illustration of horizontal semicircular canals viewed from above, deflection of cupula by angular acceleration (change in angular velocity, and change in neural activity during cupula displacement. Note spontaneous neural activity (resting level)* (CF. LOWENSTEIN & SAND, 1940).

The otolith structures of each inner ear lie in two relatively large chambers, the utricle and the saccule. These structures are thin, slightly curved, gelatinous pads with surfaces of calcium carbonate crystals. The specific gravity of the otolith crystals is between 2.71 and 2.93 (2.71 in man), considerably greater than the endolymph which is 1.0033, determined in pigeon at body temperature of 40° C (Money, Sokoloff, & Weaver, 1966). Hence, judging from mechanical properties, these organs are responsive to *linear* acceleration and to position changes relative to gravity. The size of individual otolith crystals varies in mammals between 10 and 1,000 micra, but in fish individual stones may be as great as 20mm in diameter (Groen, 1956). The otolith pad in the utricle lies approximately in the horizontal plane when the head is upright, while that of the saccule is approximately vertical. Hairs project into the gelatinous substance from cells at the base of the pad, and shearing motion of the hairs is transduced by the cells into neural impulses which signal changes in orientation relative to gravity. This system is believed to be relatively ***insensitive*** to ***angular*** acceleration per se, ***but sensitive*** to *linear* acceleration. However, this may vary according to species; e.g., consider the possible responses of the 20mm stones in Latimera (Carlstrom, 1963).

FUNCTIONAL ASPECTS OF THE NONAUDITORY LABYRINTH

Semicircular Canals

Lowenstein and Sand (1940) have shown that many nerve fibers at the base of the cupula have a spontaneous discharge rate of about 20 impulses/sec so that there is a continuous discharge from the vestibular nerve in the absence of stimulation. When the cupula in the horizontal canals moves toward the utricle, the discharge rate increases. Movement of the cupula in the opposite direction produces a decrease in the rate of firing. This is illustrated in figure 8–2. For the vertical canals, cupula deflection toward the utricle yields a decreased rate of neural firing, and deflection away from the utricle yields an increased rate.

Hence, when the head rotates with the horizontal canals in the plane of rotation, there will be a "push-pull" input to the central nervous system with an increased rate of discharge coming from one side and a decreased rate of discharge from the other side. The dual mode of response has the advantage that a single cupula can signal *either direction* of rotation within the plane of its canal in the event that one ear is nonfunctional. If one ear is suddenly lost, a difference in activity level from the two ears is thus created by virtue of the continuing spontaneous discharge from the intact ear, and disorientation and dizziness result. This continual discharge from the remaining intact ear is eventually compensated for by the central nervous system. After compensation, there is little or no difference in sensitivity to clockwise and counterclockwise acceleration (Groen, 1956; Money & Scott, 1962). After recovery, if the second ear is lost, nystagmus with fast phase toward the first ear will appear. This is called Bechterew's nystagmus and is a result of CNS compensation to loss of the first ear which is no longer balanced by the spontaneous input from the second ear. Eventually, Bechterew's nystagmus will dissipate as the CNS again adjusts to the changed state of spontaneous peripheral inflow.

When the skull is fixed in various positions relative to the plane of rotation, the three canals of each ear are stimulated differently, but the inputs from the different canals are resolved in the central nervous system such that the plane of nystagmus follows closely the plane of stimulation.

During angular acceleration, illustrated in figure 8–3, as the skull rotates to the left, the cupula would be deflected from the rest position (line 1) and the eyes would tend to remain fixed relative to the earth by a slow rotation to the right relative to the skull. However, the eyes are restricted in total excursion so that the slow drift is systematically interrupted by a fast recovery movement, creating the sawtooth pattern shown in line 4 of figure 8–3. This oscillation of the eyes, with a fast and slow component, is called nystagmus and is a typical reflex action of the eyes to semicircular canal stimulation. Electrodes placed at the outer canthi of the eyes detect horizontal eye movement by virtue of the fact that the cornea of the eye is electrically positive with respect to the retina. Hence, as the eyes turn to the right, the electrode near the right eye becomes positive, while the electrode near the left eye becomes negative, relative to an indifferent electrode. This difference can be amplified and used to drive a pen which permits recordings of eye movement.

The slope of the slow phase is usually measured and taken as an index of the intensity of the responses as shown in the 4th line of figure 8–3. During angular acceleration the sensation of rotation also increases until a constant velocity is maintained. At this point where angular acceleration ceases and a constant rotation rate is maintained, the cupula, by virtue of its elasticity,

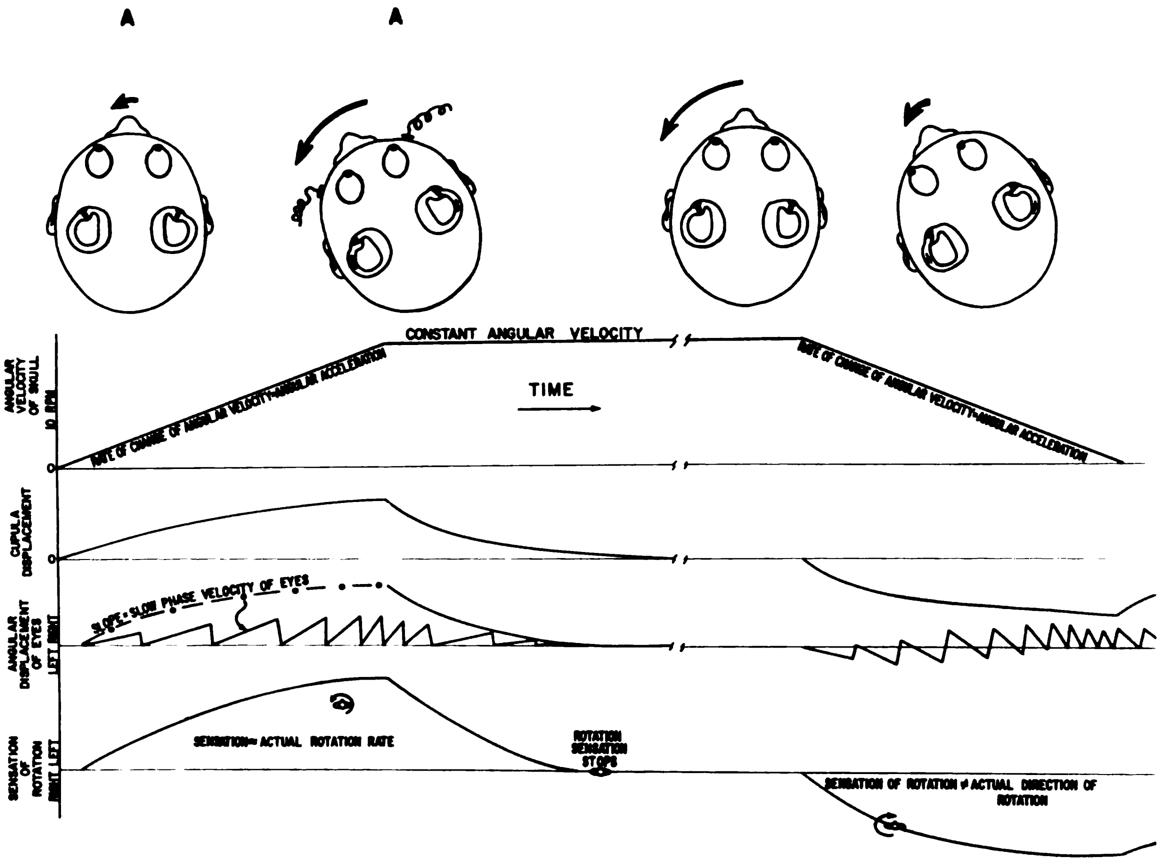
A
A
ANGULAR VELOCITY OF SKULL
10 RPM
0
RATE OF CHANGE OF ANGULAR VELOCITY=ANGULAR ACCELERATION
CONSTANT ANGULAR VELOCITY
TIME
RATE OF CHANGE OF ANGULAR VELOCITY=ANGULAR ACCELERATION
CUPULA DISPLACEMENT
0
ANGULAR DISPLACEMENT OF EYES
LEFT RIGHT
SLOPE=SLOW PHASE VELOCITY OF EYES
SENSATION OF ROTATION
RIGHT LEFT
SENSATION=ACTUAL ROTATION RATE
ROTATION SENSATION STOPS
SENSATION OF ROTATION ≠ ACTUAL DIRECTION OF ROTATION

FIGURE 8–3. *Illustrating eye movement, placement of active recording electrodes, and relation between angular acceleration, angular velocity, cupula displacement, nystagmus, and the sensation of rotation.*

commences a slow return to its null position. In about 30 to 40 seconds the cupula theoretically returns to rest and at this point the primary nystagmus and the sensation of rotation terminate. When rotation is stopped after an interval of constant angular velocity, the cupula is deflected in opposite direction by the angular acceleration of opposite sign involved in the stop. Hence, the nystagmus and the sensation of rotation during the stop and after the stop (30 to 40 seconds after the stop) are of opposite sign. Thus, during deceleration, the person in darkness has a strong sensation of rotation in one direction while he is still rotating in the other direction; and this false sensation and the inappropriate nystagmus continue for some time after the stop. (A target light fixed in front of the observer seems to rotate with the observer during and after deceleration as well as acceleration, and this series of experiences has been called the oculogyral illusion (Graybiel & Hupp, 1946). Note that this inappropriate set of responses occurs as a result of "unnatural" stimulation, i.e., angular acceleration followed by fairly prolonged constant rotation. In "natural" movements, deceleration follows acceleration closely, and hence, the deflected cupula is returned to its null zone by the deceleration without appreciable aftereffects.

Otoliths

The otoliths are usually classified as receptors for static (immobile) equilibrium. However, the fact that they are mechanically adapted to detect angular position relative to gravity does not deny that they also are capable of signaling during *change* in position relative to gravity. Lowenstein and Roberts (1950), from recordings of peripheral neural activity in the ray, found evidence for position-sensing elements and also for otolith elements which responded to change in position. Gualtierotti and Alltucker (1966), also from neurophysiological records, have concluded that the otoliths respond very quickly to any change in position; and deVries (1950), from X-ray photographs of otoliths in fish, concluded that the mechanical response of the otoliths to change in position is rapid. It is quite possible that the otolith system serves a dynamic as well as static function in supplementing information from the semicircular canal system. During rotation, the otoliths may serve to define the orientation of the plane of motion relative to gravity while the canals signal angular velocity within the plane.

EVALUATION OF PILOT DISORIENTATION

Disorientation is an incorrect mental impression of the position, attitude, or movement of the pilot and his aircraft relative to some external reference system. Usually, this reference system is the earth. Awareness of disorientation may occur only after the pilot has compared his perceived orientation with an instrument check or with direct visual contact.

Disorientation may result from (1) inattentiveness to an uncomplicated smooth flight pattern, (2) a gradual change in aircraft attitude which is below the sensory or perceptual threshold, or (3) a failure to integrate information from a complex or rough flight. Any of these could produce an error in estimation of flight attitude or position which would be experienced as disorientation as soon as an instrument or visual contact check was made. In addition, unusual force environments in aircraft can produce disorientation resulting from misleading sensory information, e.g., the experience of spinning in opposite direction after the actual spin of the aircraft has been stopped. This is a predictable response coming primarily from the semicircular canals and it is studied in the laboratory as the oculogyral illusion (Graybiel & Hupp, 1946). The pilot may feel tilted when he is not, or he may feel straight and level when he is tilted because the direction of the resultant G–vector more or less determines the perceived vertical; and this resultant vector is frequently tilted relative to the earth-vertical in typical aircraft maneuvers. This has been studied in laboratory centrifuges as the oculogravic illusion (Graybiel, 1952). If head position is changed during a turning maneuver, an illusory motion of the aircraft may be experienced due to the Coriolis vestibular reaction (Guedry & Montague, 1961). The combined movements of the head and aircraft create an inertial torque from Coriolis accelerations which stimulate the semicircular canals.

In spite of the emphasis placed on vestibular function in flight, the dominant factor contributing to disorientation in aircraft is the reduction of visual detail by darkness, rain, snow, or fog. Disorientation rarely occurs without reduced vision (Clark & Graybiel, 1955), but even vision can be misleading in flight. The most commonly listed visual illusions are autokinesis (apparent movement of a fixed light in darkness), reversible perspective (e.g., an aircraft may appear to be departing when it is approaching), and false horizons from clouds, et cetera.

Sensory information is used by the pilot in relation to his experience. The comparison of expected sensory information, deriving from the fact that a maneuver is voluntarily initiated, with actual sensory influx produced by the movement itself, determines, partially at least, spatial orientation in flight. The pilot's expected sensory information depends upon stored information on position, attitude and motion relative to the earth, and on what visible objects can reasonably be expected to move. Any intended maneuver of the plane leads to expected information and, as the movement occurs, to actual sensory information. Expected information is dependent upon the pilot's past experience in the aircraft. The plane's response depends upon its aerodynamic properties and upon external flight conditions. Thus, the plane's attitude, speed and flight characteristics, and the weather conditions control the sensory information which is fed back to the pilot as he initiates maneuvers. The *alert* pilot is aware of these factors. When the expected sensory experience and the actual sensory feedback relevant to spatial orientation are discordant with one another, the pilot experiences confusion or disorientation.

Every pilot has episodes of disorientation in various environments. He may accept these as normal or believe them to be symptoms (rightly or wrongly) of abnormality in himself or in his plane. Whether he reports disorientation, even under direct questioning, is influenced by:

1. His recognition that he was disoriented.
2. His ability to assess potential dangers in such episodes and his willingness to act on his knowledge.
3. Social and economic pressures:
    a. Will his admission have desired consequences, e.g., a medical excuse to give up a feared career?
    b. Will his admission have undesired consequences, e.g., grounding, loss of pay, status, career.
4. His rapport (or lack of it) with those to whom he might turn for help, e.g., the Flight Surgeon.

Finally, the evaluator of such a report must himself be familiar with the force environment concerned in order to make a decision as to the episode's "normality" and implications.

History

Part of the approach recommended by Jongkees (1963) for the treatment of Ménière's disease is also appropriate for evaluation of pilots: "The doctor has to show the patient that he is interested. The anamnesis has to be very thorough, not only to exclude all those other ailments which may cause spells of vertigo, but also to show the patient that his complaints are taken seriously, that *he* is taken seriously. Too often the doctor hears the word vertigo and has his prescription ready without giving the patient the opportunity to free his soul from the many things which oppress him . . . ."

Taking a careful history of a pilot who has reported disorientation is one of the most important steps in his examination. It is first necessary to establish clearly whether or not the occurrence of spatial disorientation in the pilot is due to a natural response to an unusual flight condition. The absence of similar reports by others in the aircraft does not by itself constitute evidence of an abnormal reaction from the pilot. Crewmembers could have been equally disoriented without awareness of the fact because awareness of spatial disorientation frequently depends upon checking the perceptual event against information from the instrument panel or from direct visual contact.

It is desirable to differentiate vestibular disorientation from visual illusions. According to Benson (1965), the latter account for less than five percent of the incidents reported in the Royal Air Force, but the possibility of visual origin must be checked.

In attempting to relate the disorientation to flight conditions, it is valuable to determine whether or not one of the following conditions was present: (1) reduced visual contact, (2) flickering light from sunlight on propeller blades or other sources, (3) false horizons such as might occur from cloudbanks or a line of lights at night, (4) visual illusory phenomena such as reversible perspective, autokinetic movement of lights, (5) landing in a crosswind where the line of flight of the aircraft and the heading of the aircraft do not correspond, (6) rapid changes in altitude, either climbing or diving, which might have produced pressure vertigo due to blockage and clearing of the middle ear, (7) a prolonged bank or bank and turn which sometimes produces the sensation of banking in opposite direction upon return to straight and level flight ("the leans"), (8) cessation of prolonged spinning and rolling maneuvers which can produce the false sensation of spinning in opposite direction (the oculogyral illusion), (9) very rapid spinning or rolling which can produce blurring of vision, (10) head movements relative to the aircraft during a bank or spinning maneuver (the Coriolis vestibular reaction), (11) an increase or decrease in speed, or a slip or skid which can produce errors in attitude estimation (the oculogravic illusion), (12) heavy intake of alcohol or other drugs which can produce positional nystagmus and an increased tendency toward disorientation, (13) lack of flight practice which can produce loss of habituation to flight maneuvers and heightened vestibular reactions at a time when the pilot may also be out of practice in the use of instruments, (14) mixed instrument (IFR) and contact (VFR) flight.

With the exception of item 6, the environmental factors listed above can contribute to disorientation in pilots with normal vestibular function. In regard to item 6, if a suspicion of pressure vertigo is present, the pilot should be warned that any sign of a cold or upper respiratory infection (or other disorders which disturb pressure equalization in the middle ear) may result in another episode (cf. Lundgren & Malm, 1966). The danger of pressure vertigo is that it occurs suddenly, may persist for 10 to 60 seconds, and is apt to occur at a critical time in flight, i.e., when the plane is either gaining or losing altitude rapidly. In regard to item 2, flickering light can be very annoying to normal pilots, but extreme reactions may indicate photic driving of epileptic tendencies, and this possibility should be checked.

It is also important to distinguish between a psychological disturbance as opposed to a vestibular disorder. According to Benson (1965), a number of disorientation incidents devoid of a consistent pattern are suggestive of a psychological disturbance, and he recommends a thorough investigation of the psychological disturbance which may correct the source of the disorientation. However, the presence of psychological overtones does not deny the presence of vestibular disorder. Both may be present, and of course, a severe disorientation episode may lead to psychological manifestations.

Occurrence of true vertigo in circumstances other than flight or a persistent pattern of disorientation (such as spinning or tilting) suggests labyrinthine disease. According to DeWeese and Saunders (1964), the sensation of true whirling or turning (whether of the body, or of the body and surroundings, or of surroundings) usually indicates disturbed function in the end organ, the eighth nerve or the vestibular nuclei; other sensations which may be vaguely described as dizziness suggest the presence of lesions at higher level in the nervous system. It is important for the pilot who has experienced a severe disorientation episode of organic origin to know that, in some disorders, the attacks may recur suddenly after apparent remission of symptoms. Hence, diagnosis and treatment are important. DeWeese and Saunders (1964) and also Benson (1965) provide a helpful description of the important points which should be covered in taking the history, and both emphasize that the history is perhaps the most important part of the examination.

Examination

Before specialized vestibular studies are made, a general physical examination should be given including neurological and otological examination and audiometric tests. However, as Benson (1965) has pointed out, most complaints of true vertigo, other than those of flight, are a result of labyrinthine disease. Cawthorne (1956) found labyrinthine disorders in 89 percent of vertigo cases; lesions of the central nervous system and a variety of other causes accounted for the remaining 11 percent.

Typically, a patient would be tested for vestibular imbalance as shown by nystagmus (spontaneous or positional) with fast phase in one direction and past pointing and falling (with eyes closed, feet together—the Romberg test), in the opposite direction. A refined version of standing and walking tests is currently under investigation (Graybiel & Fregly, 1965).

The semicircular canals can be stimulated by introducing fluids (water, air, ethyl chloride, carbon dioxide) into the external auditory canal. If the fluid temperature differs from body temperature, the temperature difference will be conducted to a sector of the semicircular canals. Endolymph in this sector will differ in density from the remainder of the endolymph. If the plane of the semicircular canal is alined with gravity, this density difference will cause the endolymph to fall, if the fluid is cold, or to rise, if the fluid is warm. To position the horizontal canals for precise caloric testing, a line from the outer canthus of the eye to the tragus should be vertical. Since the caloric stimulus can produce convection currents which will rotate the endolymph in either direction, each ear can be tested independently for both "right-beating" and "left-beating" nystagmus. According to DeWeese and Saunders (1964), if the head is inclined 30° forward, so that the vertical canals are alined with gravity, a "cold" or "hot" caloric irrigation will stimulate the vertical canals, and a rotatory nystagmus, i.e., nystagmus in the frontal plane, should be produced.

The eyes would be viewed with the patient wearing Frenzel lenses (20-diopter glasses over both eyes). This permits a better view of nystagmus by the examiner and also increases the intensity of nystagmus since the lenses blur the vision of the tested person. The results of the tests are interpreted as normal, hyperactive, hypoactive, or absent (DeWeese & Saunders, 1964).

However, if both hot and cold irrigations are used in each ear, then further interpretations are possible, and these are outlined by Cawthorne, Dix, Hallpike, and Hood (1956) and others (Carmichael, Dix, & Hallpike, 1956; Fitzgerald & Hallpike, 1942). A 40-second irrigation of each ear with at least 250ml of water is used (Cawthorne et al., 1956). Water temperatures used are 30° C (86° F) and 44° C (111.2° F), i.e., 7° C below and above body temperature. The patient is placed in a reclining position with the head oriented to place horizontal canals in the earth-vertical plane. The gaze is directed straight ahead and the duration of nystagmus is timed. The patient does not wear Frenzel lenses in the procedure outlined by these authors (Cawthorne, et al., 1956; Fitzgerald & Hallpike, 1942). If nystagmus of a particular direction, irrespective of the ear stimulated, is short in duration (as compared with the other nystagmus direction), this is referred to as directional preponderance and is regarded as indicative of either labyrinthine or central nervous system malfunction. Reduced response in a particular ear, irrespective of nystagmus direction is considered indicative of canal paresis; and, of course, combinations of canal paresis and directional preponderance may yield different ratios of the "hot" and "cold" caloric responses from the two ears (cf. Cawthorne et al., 1956, p. 133). Another method of attempting to quantify the caloric test was proposed by Kobrak (1918) who used short, thermally graded stimuli to estimate thresholds, and a variation of the threshold method has been proposed (McLeod & Meek, 1962).

The Bárány rotation test was once widely used (Bárány, 1907), but both ears are stimulated simultaneously during this test, and for this reason it was replaced in most clinics by caloric tests. Van Egmond, Groen, and Jongkees (1949) revived interest in rotation tests by introducing nystagmus and sensation tests which are referred to as a cupulometric examination. These tests are based upon the theoretical mechanics of the cupula-endolymph system as discussed by van Egmond et al. (1949) and further elucidated elsewhere (cf. Cawthorne et al., 1956, pp. 136–137). The logarithm of stimulus intensity is plotted with respect to the duration of the response elicited, and the resulting curve is called a sensation or nystagmus cupulogram, depending upon the response criterion selected. The cupulogram consists of a set of data points which often can be fitted approximately by a straight line. The slope of this line provides a single value which may be used in quantitive comparison of responses in clinical cases (deWit, 1953), motion sickness cases (deWit, 1953), and for comparison of pilots in different degrees of flying practice (Aschan, 1954). If the slopes for two response directions are significantly different or if the two curves are parallel but significantly displaced, there is a possibility of directional preponderance which should be further verified by caloric testing.

Improvement of commercially available rotating chairs which control angular acceleration and angular velocity, and of systems for amplifying corneoretinal potential and recording nystagmus has made it possible to get the equivalent of nystagmus cupulograms from a single vestibular stimulus of known magnitude and duration (rather than from the more time-consuming series of impulse stops from different velocities as in van Egmond's cupulometric procedure).[1]

Rotation tests offer some advantages over the caloric tests despite the fact that both labyrinths are stimulated simultaneously, and rotation tests may still be valuable as an adjunct to the caloric test. A good rotating chair permits programming the magnitude and duration of the mechanical stimulus to the canals, and thus the stimulus can be exactly determined. With careful positioning of the head relative to the plane of rotation, specific pairs of canals can be stimulated. Results must be evaluated relative to any preexisting positional or spontaneous nystagmus, and if nystagmus is being recorded, direct observation of the eyes at some point in the procedure must supplement the recordings because certain planes of nystagmus, e.g., rotary nystagmus, are not indicated with fidelity by most recording methods. However, different pairs of canals can be stimulated quantitatively by rotatory stimulation, and additional information can be gained.

Because there is an increased interest in recording and quantifying nystagmus for clinical evaluations, it is appropriate to point out experimental findings which demonstrate factors, other than the intensity and duration of the vestibular stimulus, which influence nystagmus.

Visual stimulation which provides an opportunity for still fixation inhibits vestibular nystagmus greatly (Guedry, Collins, & Sheffey, 1961). Even a point source of light, fixed relative to the patient, will reduce vestibular nystagmus markedly. Use of 20-diopter lenses to blur vision causes some reduction in nystagmus as compared with the more vigorous nystagmus which will be recorded in complete darkness. Nystagmus recorded with the patient's eyes open while in darkness is usually more vigorous and of better quality than nystagmus recorded with the eyes closed in darkness (Guedry & Montague, 1961, p. 492). Hence, one of the most sensitive ways to detect nystagmus, whether it be spontaneous, positional, or due to an adequate vestibular stimulus, is to record nystagmus with a sensitive method while the patient is in darkness with his eyes open and mentally alert. Many cases of spontaneous or positional nystagmus will be detected in this way which would otherwise be missed (Jongkees, Maas, & Philipszoon, 1962).

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[1] The logarithm of the nystagmus slow phase velocity can be plotted with respect to time starting from the point of stimulus termination and ending with the termination of the response. If y is the slow phase velocity at any time t and the initial slow phase velocity of nystagmus is y_0, then the theoretical equation which describes the decline of nystagmus is $y = y_0 e^{-\frac{\Delta}{\pi} t.}$ Taking $\log_e$ of both sides, we have: $\log_e y = \log_e y_0 - \frac{\Delta}{\pi} t$.

The slope parameter of this equation is $-\frac{\Delta}{\pi}$ and its reciprocal, $-\frac{\pi}{\Delta}$, is theoretically the same as the $-\frac{\pi}{\Delta}$ determined by van Egmond's procedure. Sensation cupulograms also could be determined by requiring estimates of subjective velocity throughout a vestibular response (cf. Cawthorne et al., 1956, pp. 139–140), but subjective velocity estimates, by most of the procedures used, require practice and instruction; and without training, the practical value of these estimates for individual evaluation is questionable.

Another factor which influences nystagmus is the subject's state of alertness. A relaxed subject in a near-sleep or reverie state may exhibit little or no nystagmus and his response can be suddenly transformed into a clear prolonged nystagmus by introducing mental arithmetic or some other task which demands mental activity (Collins, 1964; Collins & Guedry, 1962). Recently Benson, Goorney, and Reason (1965) have demonstrated that the manner of instructing the test person can influence nystagmus significantly. With several repetitions of a mild stimulus, as would be used in testing for directional preponderance, the nystagmus response may be reduced as a result of a natural tendency to relax as familiarity with the test situation develops. There is a considerable amount of evidence which demonstrates that nystagmus declines with repetitive stimulation. It is quite possible that the significance of this mental factor is minimized in the more typical clinical situation in which the patient has vision although it may be blurred by the lenses, and in which the patient is aware of the presence of the examiner who is viewing his eyes. If the stimulus is distinctly unpleasant, e.g., very cold or hot water, the probability of relaxation is further reduced. However, the attitude and actions of the examiner could influence the response, and if the more sensitive method of recording nystagmus in the dark is to be used for quantitative purposes, then it is quite important to introduce continuing mental tasks to minimize inadvertent changes in states of alertness.[2]

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[2] Several other factors influence nystagmus recorded by the corneoretinal potential technique: (1) Corneoretinal potential is influenced by visual adaptation to changes in illumination. Hence, calibration of eye movements with measured visual angles between fixation points should be interposed as frequently as is practical. (2) Eyelid potentials introduce artifacts which particularly distort the vertical component of eye movement records. These are recognizable with experience and can be greatly reduced or eliminated simply by telling the patient not to blink. (3) Placement of electrodes is important to prevent "cross-talk" between recording channels and to permit a distinction between purely vertical and purely horizontal eye movements. To detect the vertical component of eye movements, electrodes above and below the eye should be vertically alined with the pupil as the test person

With care taken in regard to the above points, vestibular nystagmus can be recorded and quantified. The practical value of clinical nystagmography, as it is frequently named, depends upon the experience of the examiner, but no less so than does the direct clinical observation of the eyes. It has the advantages of sensitivity and objective records, which can be examined quantitatively or qualitatively and reviewed at a later date. However, it has the disadvantage of being time-consuming, and its added sensitivity meets the logical enigma which is encountered by any new diagnostic tests, viz., the basis upon which it is validated depends in part at least upon the diagnostic procedures which it is to replace.

Jongkees, Maas, and Philipszoon (1962) recorded nystagmus from 341 patients with vertigo. With corneoretinal potential recordings, they detected spontaneous or positional nystagmus in 230 patients, and the ordinary clinical procedure of visual inspection with patients wearing Frenzel lenses had only detected these responses in 52 of the patients. Jongkees et al. found that only a few of the patients who did not have spontaneous or positional nystagmus showed a directional preponderance. They concluded that when spontaneous nystagmus is detected, the determination of directional preponderance will "seldom offer new aspects." Jongkees et al. (1962), Kiørboe (1959), and Stahle (1958) found a low percentage (one to three percent) of spontaneous nystagmus among normal subjects, and from this, Jongkees et al. conclude that when a spontaneous or positional nystagmus is "found in a patient, there is an organic basis for his complaints." However, the author and others (Aschan, 1954; Bergstedt, 1961) have found nystagmographic evidence of spontaneous nystagmus or positional nystagmus in from 10 to 25 percent of "normal" subjects, and this raises the question as to whether these apparently healthy individuals have some organic problem of practical significance. With sensitive electronystagmographic recordings (and more sensitive

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gazes ahead at a distant point. The horizontal component of eye movements is detected by electrodes placed symmetrically at the outer canthi. The skin should be cleansed to remove oils but with care to avoid skin abrasion. Skin abrasions seem to cause bioelectric interference, but uncleansed skin has a high electrical resistance. (4) An EKG amplification and recording system with high gain can be used. The system should have a high input impedance (a megohm or more), differential push-pull input, a time constant of at least 1.5 seconds and a frequency response with an upper cutoff of not less than 40 cps. Although a short time constant will stabilize the baseline, it also distorts the recorded velocity of the slow component of nystagmus. If the upper frequency cutoff is low, this will eliminate muscle artifacts, 60-cycle interference, and other unwanted "hash" on the record; but it also will degrade the recording of high-velocity nystagmus. Clean electrical connections between the subject's electrodes and the recorder are essential. The reference electrode should be grounded through the recorder without other inadvertent ground connections which may be present if the subject touches metal.

methods are available), it is likely that a significant degree of spontaneous nystagmus will have to be established. When recordings are used to determine directional preponderance, some differences in response between either directions of response (preponderance) or between ears (canal paresis) will be present in most people. The question is, "What is the magnitude of preponderance or positional nystagmus or spontaneous nystagmus which is within the range of normal variation?"

Jongkees et al. have proposed a solution to this problem as follows: If letters are assigned to the successive irrigations in a caloric test as follows:

a = left/cold (30° C)
b = right/cold (30° C)
c = left/hot (44° C)
d = right/hot (44° C)

the difference in excitability between ears is calculated according to the following equation:

$$\frac{(a+c)-(b+d)}{(a+b+c+d)} \times 100 \text{ percent} \tag{1}$$

Directional preponderance is calculated according to the following equation:

$$\frac{(a+d)-(b+c)}{(a+b+c+d)} \times 100 \text{ percent} \tag{2}$$

Either duration of the recorded nystagmus or maximum slow-phase eye velocity from tests a, b, c, and d may be substituted into the above equations. With 47 normal subjects, duration gave a mean difference between ears of −0.8 percent with a standard deviation of 6.7 percent. When maximum eye speed in tests a, b, c, and d were used in equation (1), there was a mean difference between left and right ears of +0.8 percent with a standard deviation of 7.5 percent. For directional preponderance, i.e., equation (2), duration of nystagmus gave a mean difference of —4 percent with a standard deviation of 15 percent; and maximum eye velocity gave a mean difference of —1.1 percent with a standard deviation of 8.7 percent. Using these statistics, Jongkees considers a left/right ear difference or a directional preponderance abnormal when the obtained values exceed double the standard deviation from the mean. With this criterion in evaluating 351 patients with vertigo, Jongkees concluded that the mean slow-phase eye velocity was a more critical measure than duration of nystagmus in detecting symptoms of vestibular disorder, and that electronystagmography is indispensable in clinical practice in diagnosing disability.

For caloric tests where nystagmus is not recorded but is directly observed without use of Frenzel lenses on the patient, Cawthorne et al. (1956) have also provided a statistical analysis of the normal occurrence of left/right sensitivity differences and directional preponderance. They measured duration of nystagmus and used the numerators only of equations (1) and (2) (in other words, they did not use percentage scores) to estimate differences in response. With this method of statistical treatment and 93 normal subjects, they found a mean difference in sensitivity between left and right canals (numerator of equation (1)) of —1.93 sec and a standard deviation of 15.5 sec. For directional preponderance (numerator of equation (2)), they found a mean value of —0.15 sec with a standard deviation of 15.7 sec.

Validity of test procedures of this type for pilot examination is further supported by examination of pilots who have had vertigo attacks. Benson (1965) has reported preliminary results of the examination of Royal Air Force pilots whose main presenting symptom was spatial disorientation. He reports that some 90 percent of those who had experienced illusory perception of either aircraft attitude or aircraft motion showed a minor vestibular abnormality such as directional preponderance in one or more of the tests administered. Two other groups of patients were similarly examined: a group whose array of symptoms suggested neurotic disorders presented a 25 percent incidence of abnormal findings, and motion sick aircrews presented an incidence of only 13 percent abnormal findings in vestibular function tests.

Tests of otolith function are not clearly established in the routine clinical examination. When the whole body is tilted relative to gravity, many structures other than just the otoliths are stimulated; and for this reason, it has been difficult to establish responses that are controlled solely by the otolith system. Measurement of ocular counterrolling appears to be a test of otolith function (Miller & Graybiel, 1963), but as it is usually carried out, it is time-consuming and may not reflect the dynamics of otolith function. Responses which have been studied as possible indicators of otolith function include perception of tilt relative to gravity, perception of tilt relative to resultant forces on centrifuges, perception of linear velocity during oscillation on parallel swings, eye movements during linear oscillation, counterrolling eye movements during static tilt, and eye movement patterns during constant velocity rotation about a horizontal axis. When results from these tests are obtained from presumably normal human subjects, they differ quantitatively from results obtained from individuals believed to be without vestibular function. Theoretically, the stimuli involved in these tests should stimulate the otolith structures and not the semicircular canals. Since the tests discriminate individuals with and without labyrinthine function, and since they are supposedly stimuli to the otoliths, they may prove useful as indicators of otolith function, but their final interpretation and practical clinical application await further experimental evaluation.

Bergstedt (1961) has proposed that positional alcohol nystagmus is dependent upon otolith function and therefore would constitute a test of the otolith system. Although people without inner ear function do not have positional alcohol nystagmus (Harris, Guedry, & Graybiel, 1962), recent experiments by Money, Johnson, & Corlett (1965) suggest that functional canals rather than functional otoliths are necessary for positional alcohol nystagmus. These findings question the validity of positional alcohol nystagmus as a test of the otolith system. Because tests of otolith function have not commonly been given, the extent to which quantitative changes in otolith function influence pilot disorientation is conjectural, but it is reasonable to expect that any fairly sudden change in otolith function due to labyrinthine disorder would have a severe disorienting effect.

Preventive Measures

Disorientation in flight will be experienced by most normal pilots. The goal is to reduce to a minimum its degradation of flight performance. Benson (1965), Jones (1957), and others (Clark & Graybiel, 1955; 1957) have summarized important preventive measures.

Training in regard to the degree of disorientation which can be experienced by normal pilots is important. Inflight instrument training is ordinarily sufficient to illustrate disorientation, but the fact that it is normal to be disoriented under some of the conditions of flight should be emphasized. A rotating chair can be used to illustrate severe disorientation and the temporary blurring of vision which strong nystagmus can produce.

Knowing when disorientation is apt to occur is helpful, and hence, flight conditions which are conducive to disorientation should be reviewed. Reduced vision, mixed contact and instrument flight, and distraction of attention during instrument flight are all conducive to disorientation. Lack of flight practice can reduce ability to suppress misleading sensory information at the same time that proficiency with instruments is reduced. The "leans" are a normal phenomenon following a prolonged bank. Very high-altitude flight can produce a horizon significantly below true horizontal flight and thus produce errors in attitude estimation (Jones, 1957). A rolling or spinning maneuver is apt to produce disorientation which may become very severe if the maneuver is continued for several turns. Jones recommends that rolling should not be continued beyond three turns and that the maximum rate of roll should not exceed 200 degrees/sec, i.e., about 0.5 rolls/sec. It is, of course, possible that exigencies of combat may demand exceptional maneuvers. Jones further suggests that directing the gaze toward the horizon rather than the earth beneath will facilitate visual suppression of the undesirable vestibular nystagmus which follows a spin. Long-term aftereffects of alcohol (Aschan, Bergstedt, Goldberg, & Laurell, 1956; Bergstedt, 1961; Jones, 1957) may increase the probability of disorientation. The rapid change of altitude in modern aircraft (including helicopters) has apparently increased the incidence of pressure vertigo (Lundgren & Malm, 1966). It has been recommended that individuals with a previous episode of pressure vertigo be temporarily grounded during colds and upper respiratory infections and that they be instructed on maintaining middle ear pressure equalization during changes in altitude (Jones, 1957). Pilots sometimes shake their heads in an effort to dispel disorientation. According to Benson (1965), laboratory tests actually confirm that this practice can reduce misleading vestibular sensations, but it is important to avoid such head movements during a rotational movement, e.g., spinning or rolling, of aircraft because of the disturbing vestibular Coriolis reaction (Guedry & Montague, 1961). In this connection, movement of the head which diverts attention from instruments or visual contact during a bank and turn should be avoided. For example, during a level turn, increased head-to-seat G can be interpreted as a climbing attitude, and a head tilt to the right, perhaps to switch radio channels, can also produce a climbing sensation from the Coriolis reaction.[3]

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[3] The Coriolis vestibular reaction (Guedry, 1965a; 1965b; Guedry & Montague, 1961) is produced when a person tilts his head about one axis, x, while he is in a vehicle which is rotating about another axis, z. The strength of the reaction is controlled by the magnitude of the angular velocity (rate of rotation) about axis z and the total angle through which the head is tilted about axis x, assuming the head tilt is made at a natural rate. If axes z and x are at right angles to one another, the reaction will take place about another axis, y, which is at right angles to both axes z and x. For example, while a chair is rotating to turn a person to his right, if the top of the head is tilted directly toward the right shoulder, the person will experience a backward tilting motion as though he is in a climbing attitude. He will also have nystagmus with fast component directed upward. This effect will occur

This combination may occur when attention has been diverted from contact flight or from the pertinent instruments. An instantaneous reaction to this false perception at low altitude could be dangerous.

According to Benson (1965) most aircrew whose presenting symptom is disorientation show no evidence of organic disease. Those who do have organic basis should, of course, receive the normal medical treatment. In cases without demonstrable organic abnormality, Benson suggests shift of flight assignment, e.g., flying dual, or flying a less stressful aircraft, to reduce stress if the disorientation seems to be associated with a particular aircraft or aircraft maneuver. Temporary or permanent grounding may be necessary. He also suggests review of causes of disorientation so that any hypochondriacal concern is alleviated for those cases in which normal disorientation is regarded as a personal defect.

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with a person on or near the center of rotation, as in a rotating chair, spinning aircraft, or tumbling space capsule; and it will also occur when the person is away from center of rotation, as on a centrifuge or an aircraft in a bank and turn.

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CHAPTER 9

Vision in Aerospace Medicine

VISION AND THE EYE

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VISION AND THE EYE

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INTRODUCTION

Since man first ventured into the air, he has depended upon his eyes to guide him in safe flight. This is just as true today as it was in the beginning, despite the introduction of a host of electronic aids such as GCA, ILS, radar detection devices, mirror and lens landing systems, onboard computers, and many others. Even in space flight, in which takeoff and landing are largely automated, such maneuvers as docking in space require excellent vision. Just as no machine has yet replaced the pilot in the cockpit, neither has a substitute been found for the eyes of the pilot. It is axiomatic, then, that a pilot needs to have vision that is adequate in every respect. He must have normal visual acuity, ocular muscle balance, field of vision, color vision, depth perception, and night vision. The Flight Surgeon must examine candidates for flight training to insure that those accepted are not only visually qualified at the time, but are also unlikely to have their vision worsen rapidly in ensuing years, which would limit the types of flying they could perform. All aviators must be examined on an annual basis and, should minor defects be noted, the Flight Surgeon will need to have knowledge of how great a discrepancy is allowable without jeopardizing that pilot's safety in flight. Flight Surgeons should also be acquainted with the more common types of ocular pathology, should know how to give initial treatment for various kinds of ocular trauma, and should know the eye hazards involved in their operational units and how to cope with them.

VISUAL ACUITY

Visual acuity is tested by the use of the Snellen Eye Chart. This chart originally evolved from the work of an English astronomer who tried to determine the "Minimum Separable." He wanted to determine how far apart two stars must be in order for them to be perceived by the normal eye as two

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stars rather than as one star, and the figure he arrived at was one minute of arc (1′). Later, Snellen used this determination to develop his eye chart. First, he took the letter "E" and constructed it of such size that each leg subtended an angle of one minute of arc at a distance of 20 feet (or 6 meters), this being the distance decided upon as representing optical infinity for all practicable purposes. Thus the whole letter "E" subtended a visual angle of 5 minutes of arc at a distance of 20 feet (see figure 9–1). Other letters were constructed of similar sizes for use on the 20-foot line, and then letters of a size to subtend visual angles of 5′ of arc at 25 feet, 30 feet, and greater distances. Visual acuity is expressed as a fraction in which the numerator is the distance of the patient from the eye chart (usually 20 feet) and the denominator is the lowest (smallest) line that the patient can read correctly. If a patient or candidate can read the 20-foot line at 20 feet, this is recorded as 20/20, and it

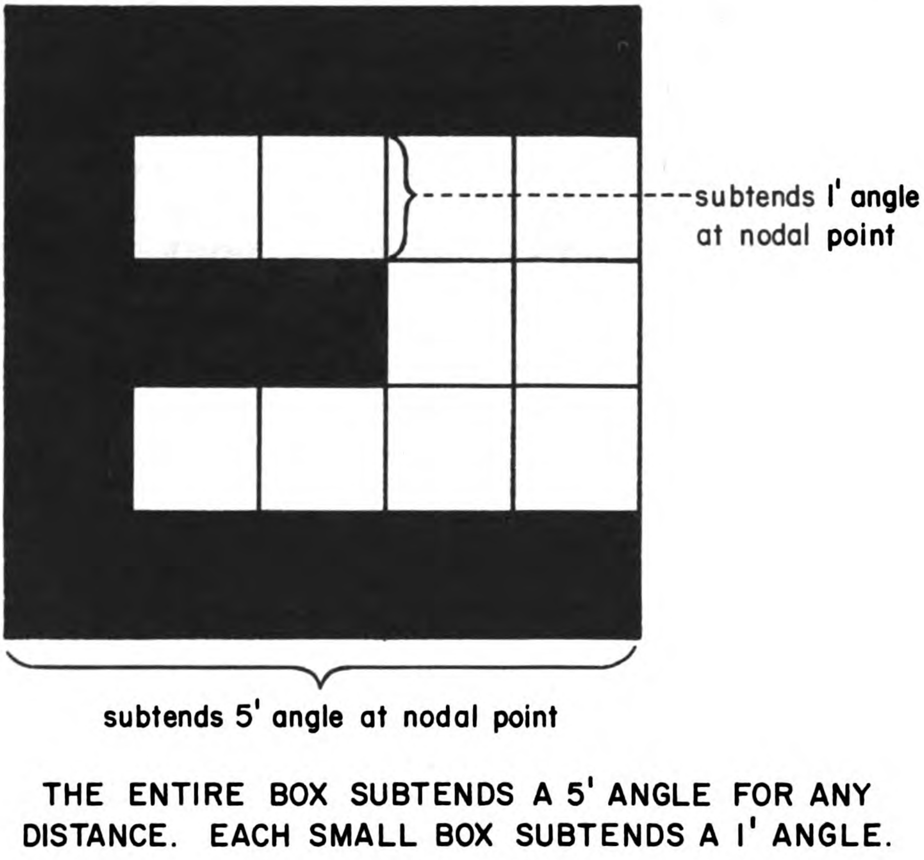

subtends 1' angle at nodal point
subtends 5' angle at nodal point
THE ENTIRE BOX SUBTENDS A 5' ANGLE FOR ANY DISTANCE. EACH SMALL BOX SUBTENDS A 1' ANGLE.

FIGURE 9–1. *Construction of a letter used in Snellen Eye Chart.*

means that the patient can see at 20 feet what the "normal" person can see at 20 feet. If he can only read the 40-foot line correctly, it is recorded as 20/40 and it means that he must stand at 20 feet to see what the "normal" person can see at 40 feet. In actuality, the "minimum separable" for most people is smaller than one minute of arc, as evidenced by the fact that many of us can read 20/15 or even 20/10, but one minute of arc is the figure upon which the system is based and it is as good as any. When a person cannot see the largest letter on the eye chart from 20 feet, he is moved closer to the chart until he can see the top letter (usually a "200-foot" letter). If this letter can be seen at 10 feet, the vision is charted as 10/200, etc. If the largest letter cannot be seen at any distance, one determines if the patient can count fingers, and records the vision as "Finger Count at 2 feet," for example, and if vision is even less than this, one determines if the patient can perceive light and if he can tell where the light is located. This type of vision is recorded as "Light Perception Only" (LP) or as "Light Perception and Projection" (LP&P).

If the subject wears glasses, visual acuity should be charted both with and without the correction. If not presently corrected to 20/20, the vision with pinhole disc (see below), or glasses plus pinhole disc, should be included as follows:

VOD 20/80 corrected to 20/40 by lens—20/20 lens plus pinhole;
VOS 20/100 corrected to 20/20 by lens.

The pinhole disc is simply a disc with a hole in it. It works like a pinhole camera and, if there is no ocular pathology, it should afford as good visual acuity as the best lens correction. It gives a quick determination of the visual potential of the eye. Perhaps you have performed a physics experiment wherein you set up a candle, a piece of cardboard with a hole in it, and behind this, another cardboard to act as a screen. You get an inverted picture of the candle on the screen because only the rays of light that pass straight through the hole reach the screen and these pinpoints of light create an image. It does not matter how close or far the screen is from the pinhole, you still get the image. Therefore, when a subject uses a pinhole disc a clear image is produced on his retina whether he is hyperopic, myopic, or astigmatic. If a patient's glasses do not correct him to 20/20 but the pinhole does, all he probably needs is a change in his correction. However, if the pinhole does not correct him and his health record shows that he previously had 20/20 in that eye, it may mean that ocular pathology is present and he is losing vision, thus constituting an emergency. The cause of the visual loss must be determined as early as possible and indicated treatment instituted.

The pinhole may be useful under field conditions. If a candidate is applying for some program which requires that his vision be correctable to 20/20 and the equipment to perform a refraction is not available, his vision should be checked with the pinhole. The information as to whether he corrects to 20/20 with the pinhole should be included in the Standard Form 88 sent to the Bureau of Medicine and Surgery.

For authoritative information concerning visual acuity standards for all programs, an up-to-date *Manual of the Medical Department* should always be consulted.

REFRACTIVE ERROR

When parallel light rays, coming from a distance, are brought to a focus upon the retina of any eye that is not accommodating, that eye has no refractive error and is said to be *emmetropic.* When parallel light rays are *not* brought to a focus upon the retina of an eye that is not accommodating, it is said to be *ametropic. Refractive error* is the lens prescription required to convert an ametropic eye to emmetropia. There are only three types of ametropia to be considered; hyperopia, myopia, and astigmatism.

Hyperopia is defined as an eye in which parallel light rays coming from a distance are brought to a focus behind the retina when the eye is at rest (i.e. not accommodating). Obviously, this cannot happen as the rays are stopped by the retina before they can reach a focus, but it is easier to visualize the process of refraction by thinking of them as focusing behind the retina. The hyperopic eye is usually a small, short eye, but in any case its refracting surfaces are not strong enough to bring parallel rays to a focus on the retina unless the eye accommodates. (See figure 9-2.)

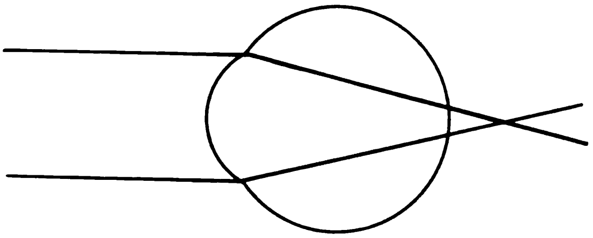


FIGURE 9-2. *Hyperopic eye.*

Myopia is defined as an eye in which parallel light rays coming from a distance are brought to a focus in front of the retina with the eye at rest. This is usually a large or long eye. In any case its refracting surfaces are too strong for its axial length. Should it try to accommodate, the focus will be brought further forward and thus further out of focus. It must try to relax as much as possible when looking into the distance. If it looks at a near object, it receives diverging rays and the focus is thereby moved back onto the retina. (See figures 9–3 and 9–4.)

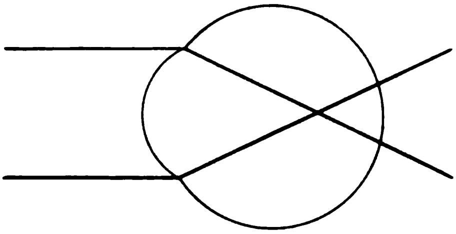


FIGURE 9–3. *Myopic eye looking at a distant object.*

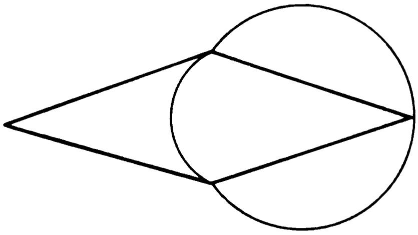


FIGURE 9–4. *Myopic eye looking at a near object.*

Astigmatism may be regular or irregular. Irregular astigmatism is seen in cases of scarred cornea, and in keratoconus where the refracting surfaces are irregular and a blurred focus results. Regular astigmatism is the type usually encountered. Here the refracting surfaces do not have the same power in all directions; however, the curvatures are regular in that there are always 90 degrees between the sharpest and flattest curves with a smooth progression between the two. This can involve either the cornea or lens or both, but usually it is the cornea. Astigmatism can best be explained by asking the reader to visualize a balloon filled with water resting upon a table. The weight of the water causes the balloon to sag so that its vertical diameter is shorter and its horizontal diameter is longer. Now visualize the curves on the end of the balloon opposite to the inflation end. Vertically the curve will be sharper (shorter radius of curvature), and horizontally the curve will be flatter (longer radius of curvature), and there will be a smooth progression between the two. This is similar to the curvatures of an astigmatic cornea (or lens). As a matter of fact, most individuals with astigmatism have the sharpest curvature in the vertical and for this reason this type is called "astigmatism with the rule." Others have the sharpest curvature in the horizontal, in which case it is called "astigmatism against the rule."

It should be noted that with any refracting surface, the sharper the curvature (that is, the shorter the radius of curvature), the more refractive power

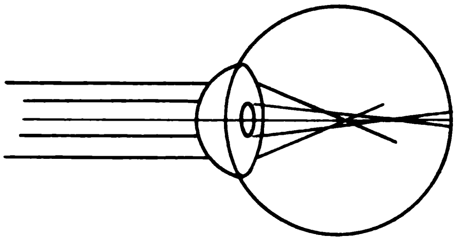


FIGURE 9–5. *Conoid of Sturm.*

it will have. When the curvatures are not the same in all directions (as they are in regular astigmatism) it is obvious that there can be no point focus anywhere. In fact, the resulting optical figure is called a "Conoid of Sturm" (as diagrammed in figure 9–5). It consists of two focal lines through which all rays entering the eye from a single point in space must pass. These are called the anterior and posterior focal lines. Approximately halfway between them is the Circle of Least Diffusion. That portion of the conoid between the two focal lines is called the "Interval of Sturm." A good mental concept of the Conoid of Sturm is essential in just about everything done in refraction. It is well to consider what the cross section of the conoid is at various points along its course. Note that when the ray enters the eye it has the shape of the pupil (round if the pupil is round). Then it begins to elongate in the direction of the anterior focal line, horizontally in the case of astigmatism with the rule (which will be explained later). It thins out until it becomes a line. Now it starts rounding up again until it becomes a circle, after which it begins elongating and thinning out in the direction of the posterior focal line. After passing through the posterior focal line it rounds up again and theoretically proceeds to infinity. (See figure 9–6.)

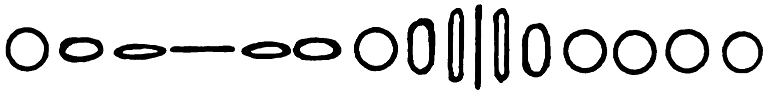


FIGURE 9–6. *Cross section of Conoid of Sturm.*

Let us now define two terms which we will use subsequently in discussion. A ***meridian*** of the eye is the same as a meridian of any other globe, that is, it is a circumferential line that passes through two opposite poles. The meridians on the earth pass through the north and south poles. Meridians of the eye pass through the anterior and posterior poles and we are principally interested only in the portions that pass over the cornea. The horizontal meridian is termed the "180 degree meridian." When facing the patient, his

5 degree meridian is above the horizontal to your right and below the horizontal to your left, progressing in this fashion to the vertical meridian, which is termed the 90 degree meridian, and so on until the horizontal or 180 degree meridian is reached again (figure 9-7). Numbering higher than 180 degrees is not done. The same notation is used for both eyes. It is worthy of mention that this terminology is used worldwide; in fact, the language of spectacle prescription is worldwide. A prescription written in the United States could be understood and filled anywhere in the world.

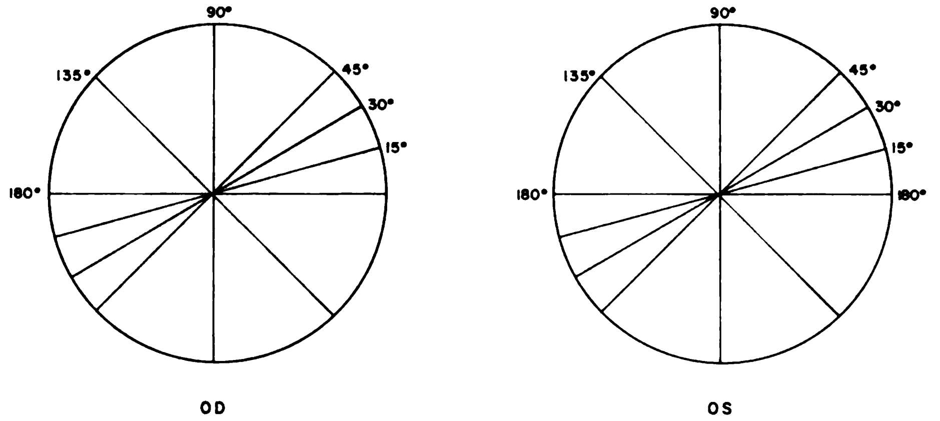


Figure 9-7. *Designation of meridians of the eye.*

Accommodation will be discussed more fully in a later paragraph, but it is defined as the ability of the eye to increase its refractive power through the action of the ciliary muscle upon the lens, making it assume a more globular shape. Action by the ciliary muscle can only increase the power of the lens. When the ciliary muscle relaxes, the lens flattens out, but the ciliary muscle cannot exert any force to make it even flatter.

The total refractive power of the eye is roughly 60 diopters, 40 of which are due to the cornea and 20 to the lens.

A further discussion of the three types of refractive error will perhaps create better understanding. Hyperopia is the condition which the layman describes as farsighted, meaning that the hyperope can usually see better at distance than near. But this is not always the case. Consider the person who is just a little hyperopic. By accommodating he can bring his focus forward and see both at distance and near depending upon how much he accommodates. If he is more hyperopic, he may be able to accommodate enough to bring parallel light rays coming from a distance to a focus on his retina, but be unable to accommodate enough to focus the divergent rays from a near point. Such a hyperope would be able to see clearly at distance but not near. If he is extremely hyperopic, he may be unable to accommodate sufficiently to bring even the parallel light rays to a focus on his retina while divergent rays from a near point would be focused even further behind his retina. This individual could not see clearly either at distance or near unless, of course, he wore glasses. Therefore, the term "farsightedness" is not truly descriptive of hyperopia.

On the other hand, "nearsightedness" is a fairly good descriptive term for myopia. Parallel light rays coming from a distance upon entering a myopic eye are focused in the vitreous and are therefore blurred on the retina. When such an eye looks at a near object, divergent rays are received and these can be focused on the retina. The myope can see nearby, but not at distance.

Total hyperopia is the amount of hyperopia that can be measured by refracting the eye while the ciliary muscle is in a state of flaccid paralysis due to the action of cycloplegic medications. Manifest hyperopia is the amount of hyperopia that can be measured by refracting the eye without the use of a cycloplegic. The manifest is usually less, and never greater, than the total hyperopia since the normal ciliary muscular tone has some effect on the lens, making it more spherical and therefore more powerful from a refractive standpoint. The difference between the total and the manifest hyperopia is the latent hyperopia. It is latent because, although not yet manifest in the young eye, normal aging will cause progressive loss of tone of the ciliary muscle and more and more of the latent hyperopia will become manifest. Most of the world's population are hyperopes of a minor degree who can see 20/20 uncorrected. More severe hyperopes require a hyperopic correction in order to read the 20/20 line. The amount of this correction is the amount of absolute hyperopia. The difference between the amount of lens correction which the patient will accept without cycloplegia (i.e., the manifest hyperopia and the amount he requires to see 20/20) is called the accommodative or facultative hyperopia. The accommodative portion of the patient's hyperopia is the portion for which he can contract his ciliary muscle and see 20/20 or relax his accommodation and accept the hyperopic correction, and still see 20/20.

It is because of the possibility of a large amount of latent hyperopia that cycloplegic refractions are performed on all applicants for flight training. These applicants are in their early twenties and have the high accommodative power of youth. They may have a high degree of hyperopia and still be able to read 20/20 as required. They may also have a very strong ciliary muscle tone with the result that much of their hyperopia is still latent. As mentioned earlier, it is highly desirable that applicants selected for flight training be not only physically fit at the time but also have a reasonable expectancy of remaining capable of continuing unrestricted flying throughout their careers. If a young man with a high degree of total hyperopia, a large part of which is latent, is allowed to slip by and enter a flight program, within a few years, much of the latent will become manifest and some of the manifest will become absolute as he loses both ciliary muscle tone and accommodative power. In other words, he will need glasses and his usefulness as a military aviator will be restricted. The only way to prevent this is by doing a baseline cycloplegic refraction on all candidates for pilot training. Plus 2.50 diopters (see paragraph on lens power below) is the limit of hyperopia that is allowable for original selection.

In myopia, there are no subdivisions corresponding to those of hyperopia but there is one condition that has some similarity to latent hyperopia—that called pseudomyopia or ciliary spasm. This condition can occur in individuals who may not be myopic at all but require a myopic correction in order to see. This is due to the fact that the ciliary muscle is in spasm, causing the lens to generate more refractive power than needed. It can also occur in myopes causing them to require more myopic correction than actually needed. Because of this, in myopia it is sometimes necessary to perform a cycloplegic refraction to be absolutely certain of the patient's refractive error. Pseudomyopia or ciliary spasm is a psychophysiologic phenomenon and frequently a psychiatric interview serves to uncover the basis of the situation, and even to correct it. It can also be treated with a few weeks of mild cycloplegia in the hope that the spasm will not return.

Astigmatism is subdivided according to the way it can be combined with either hyperopia or myopia. These are diagrammed in figure 9–8 below. Please note that liberty has been taken with spatial relationships in that it has been necessary to show horizontal focal lines as running in an anterior-posterior direction whereas they actually should run perpendicular to the sagittal plane of the eye. Also the vertical focal lines are shown in a posterior position as they are in astigmatism with the rule. The two focal lines could be reversed or could be in any meridian as long as they are 90 degrees from each other. With the eye at rest, the following types of astigmatism occur: in ***compound hyperopic astigmatism,*** both focal lines are behind the retina

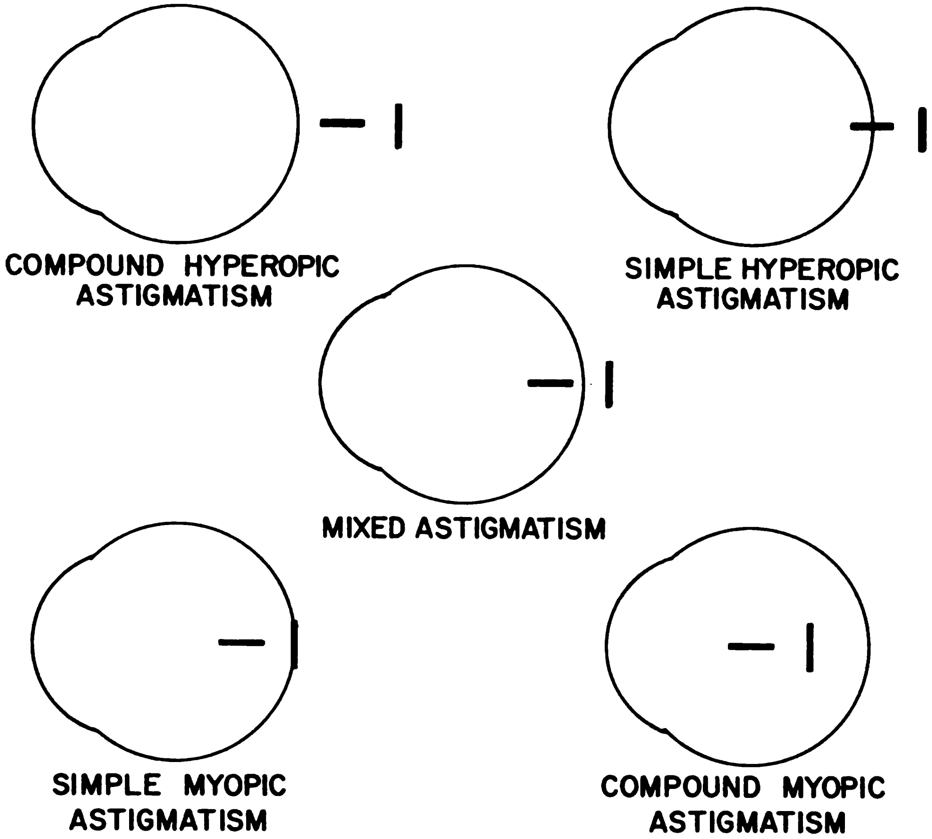

COMPOUND HYPEROPIC ASTIGMATISM
SIMPLE HYPEROPIC ASTIGMATISM
MIXED ASTIGMATISM
SIMPLE MYOPIC ASTIGMATISM
COMPOUND MYOPIC ASTIGMATISM

Figure 9–8. *Types of astigmatism.*

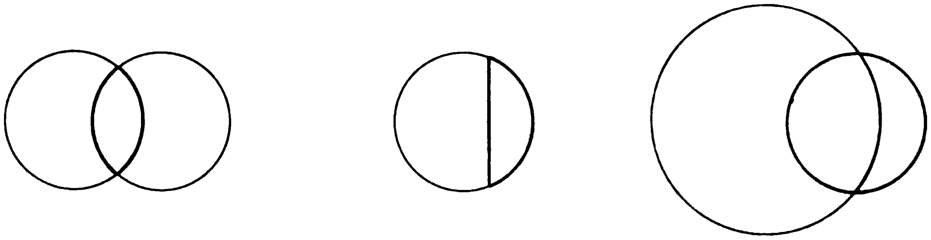
(with the eye at rest in all cases); in *simple hyperopic astigmatism,* there is one focal line behind the retina, the other on the retina; in *simple myopic astigmatism,* one focal line is on the retina, the other in front; in *compound myopic astigmatism,* both are in front; and in *mixed astigmatism,* one is behind the other and in front of the retina.

LENSES

The lenses used in measuring and correcting refractive error and in prescribing spectacles may be either spherical, cylindrical, or combinations called spherocylindroids (toric) lenses. Spherical lenses are so called because they may be thought of as either parts of spheres or "casts" of spheres. Plus spheres have plus power or converging power. Minus spheres have minus power or diverging power. A lens without power, that is, plain glass, is called a plano lens, abbreviated PL. Plus spherical lenses can be thought of as having been obtained by intersecting a sphere with a plane or with another sphere and are denoted as in figure 9–9.

Minus spherical lenses can be obtained by taking a cast of the space between a sphere and a plane, or between two spheres, or by intersecting a sphere with another sphere. These shapes are depicted in figure 9–10. It is well to note the difference between convexoconcave and concavoconvex. Convexoconcave is what remains when a sphere has been intersected with a smaller sphere. The concave surface has the smallest radius of curvative and the lens has minus power. A concavoconvex is what remains when a sphere has been intersected with a larger sphere. The convex surface has the smallest radius of curvature and the lens has plus power.

Cylindrical lenses are so called because they can be thought of as having been cut from cylinders (plus cylinders) or as having been made from casts of cylinders (minus cylinders) (see figure 9–11). Cylinders are said to have an "axis" which corresponds to the axis of the cylinders from which they were cut. Since there is no curvature in the axis, there is no power. The curvature is 90 degrees away from the axis and here lies the full power of the cylinder. At angles in between, there is partial power in proportion to the degrees



Biconvex **Plano-convex** **Concavo-convex**

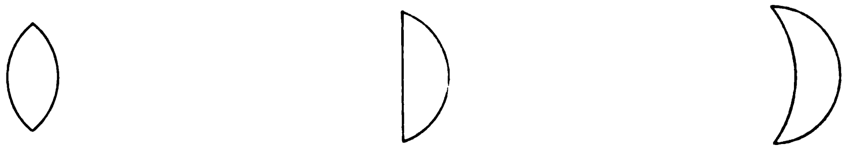


FIGURE 9–9. *Plus spherical lenses.*

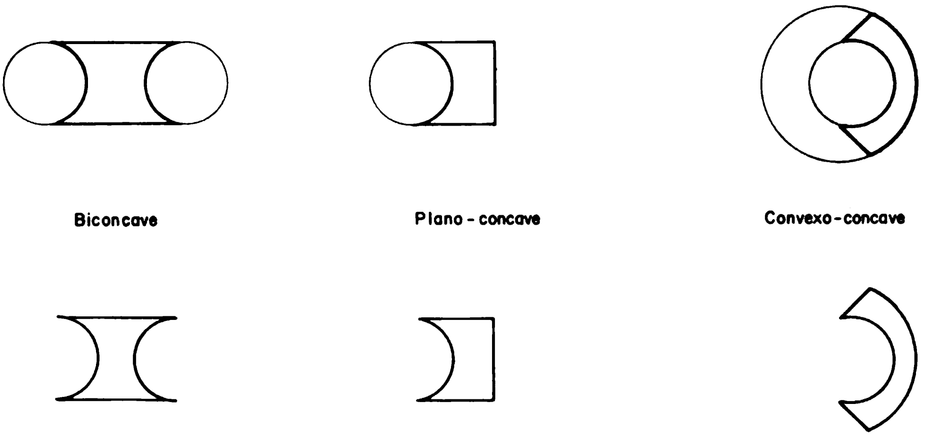


FIGURE 9–10. *Minus spherical lenses.*

that the meridian in question lies between the axis meridian (no power) and the meridian of full power.

Spherocylindroids or toric lenses are combinations of spheres and cylinders. They may be obtained in a number of ways. One could grind the back surface of a lens spherically, either plus or minus, and the front surface cylindrically, either plus or minus, depending upon the power desired, or one could make the front surface spherical and the back surface cylindrical. One surface could be plano and the other a toric surface; the toric surface could have one meridian with a certain radius of curvature and 90 degrees from that a meridian with a different radius of curvature, with a smooth progression between the two. In performing a refraction, spherocylindrical power is obtained simply by placing both a sphere and a cylinder before the patient's eye.

Lens Power

Parallel light rays passing through a plus sphere are converged and brought to a focus behind the lens at the focal point (figure 9–12). The distance from the center of the lens to the focal point is called the focal distance. The inverse of the focal distance in meters gives the diopteric power of the lens. A plus lens with a focal distance of one meter is therefore a +1 diopter lens; a plus lens with a focal distance of ¼ meter is a +4 diopter lens. These figures can also be written as +1D and +4D or as +1.00 and +4.00. The latter method is

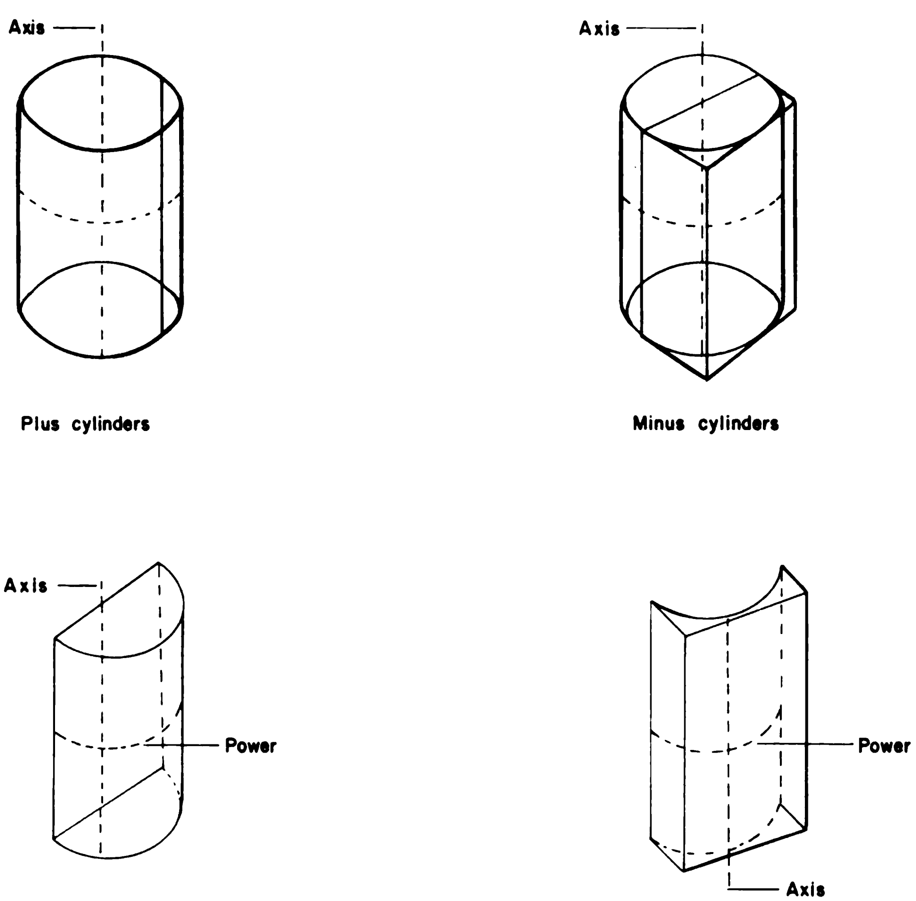


FIGURE 9–11. *Types of cylindrical lenses.*

used in writing spectacle prescriptions and two digits always appear after the decimal.

Parallel light rays passing through a minus sphere are diverged and the lines of these rays must be extended backward toward the source to find the focal point which lies in front of the lens (figure 9–13). Again, however, the distance from the center of the lens to the focal point is the focal distance and the inverse of the focal distance gives the diopteric power.

Cylindrical lenses do not have a focal point but have rather a focal line, which is parallel with the axis of the cylinder. In the case of a plus cylinder, the focal line is behind the lens; with a minus cylinder, it lies in front of the lens as with plus and minus sphere (figure 9–14). The distance from the center of the lens to the focal line is the focal distance, and the inverse of this (in meters) is the diopteric power.

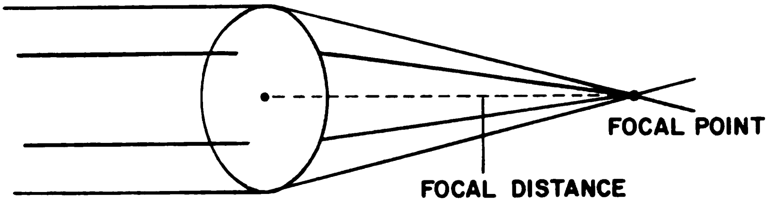


FIGURE 9-12. *Effect of plus sphere on parallel light rays.*

Toric lenses have neither a focal point nor a focal line but rather have two focal lines and, in fact, a "Conoid of Sturm." Their power must be expressed in terms of the sphere and the cylinder from which they can be thought of as having been derived, and here the meridian of the axis of the cylinder must be included. For example +1.00 —.50 x 75 means: combine a plus one diopter sphere with a minus one-half diopter cylinder with its axis at 75 degrees. In examining eyes for refractive error and in writing prescriptions for spectacles, it is customary to deal in increments of one-fourth of a diopter. One-eighth-diopter increments are also available but are seldom used. Once in a while it is necessary to use an eighth diopter (i.e. ±.12, .37, .62, etc.) in a very particular patient but for the most part this is considered "gilding the lily."

Use of Lenses

Plus spheres are used to correct hyperopia without astigmatism, minus spheres are used to correct myopia without astigmatism; plus cylinders are utilized in simple hyperopic astigmatism, minus cylinders in simple myopic astigmatism; and toric lenses are used in compound hyperopic, compound myopic, and mixed astigmatism.

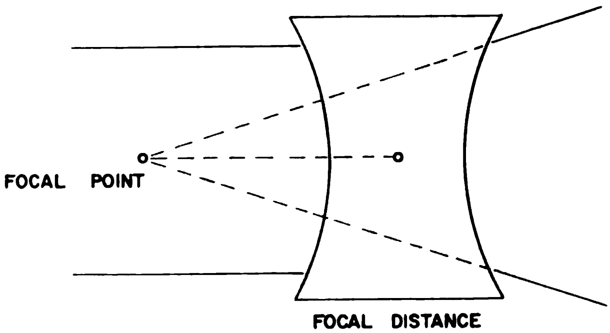


FIGURE 9-13. *Effect of minus sphere on parallel light rays.*

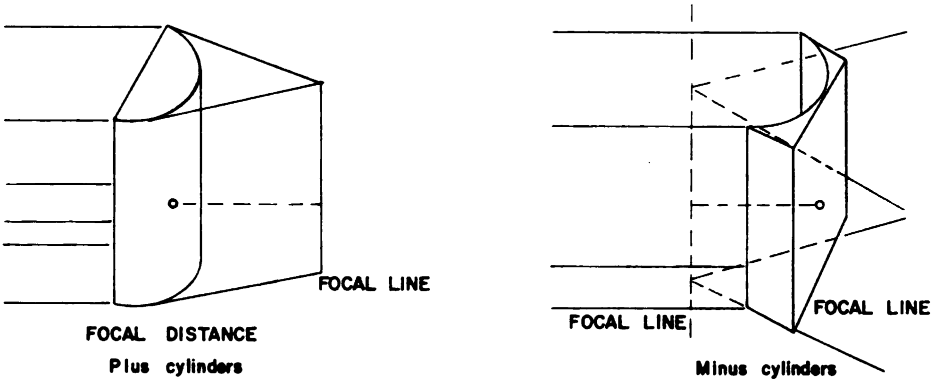


FIGURE 9–14. *Effect of plus and minus cylinders on parallel light rays.*

Transposing Lens Prescriptions

In the examples given in figure 9–15 below, cylinders of plus and minus power have been indicated. Actually either plus or minus cylinders, as desired, could be used in any particular case because *any* prescription can be written in either plus or minus form and still represent the same lens power simply by changing the power of the sphere and the axis of the cylinder. For example, in order to achieve a lens with a power of +1.00 in the horizontal and +.50 in the vertical, the following could be done. The power of lenses placed together can be added algebraically. So a +1.00 sphere could be chosen and placed with a —.50 cylinder with its axis at 180 so that its power would be at 90 (figure 9–16). The +1.00 has a power of +1D in all meridians. The —.50 cylinder has plano in the 180 and —.50 in the 90 degree meridian. Placing these two together and adding +1.00 and —.50 in the vertical gives a resultant power of +.50. Adding the power of the two lenses in the 180 degree meridian, +1.00 and PL gives +1.00 in the horizontal. These are the two powers hypothetically sought. However, a +.50 sphere could be selected and a +.50 cylinder added to it with its axis in the 90 degree meridian. Adding the two powers in the vertical meridian would result in +.50 and plano =+.50. Adding the two powers in the horizontal meridian gives +.50 and +.50 or +1.00 (figure 9–17). Again, these are the two powers desired, achieved in two different ways, one using a plus cylinder and one using a minus cylinder. It can be seen that +1.00 —.50 x 180 and +.50 +.50 x 90 represent the same resultant power, which could be written either way and would still represent the same piece of glass. An optician would fill such a prescription in the same way regardless of the way

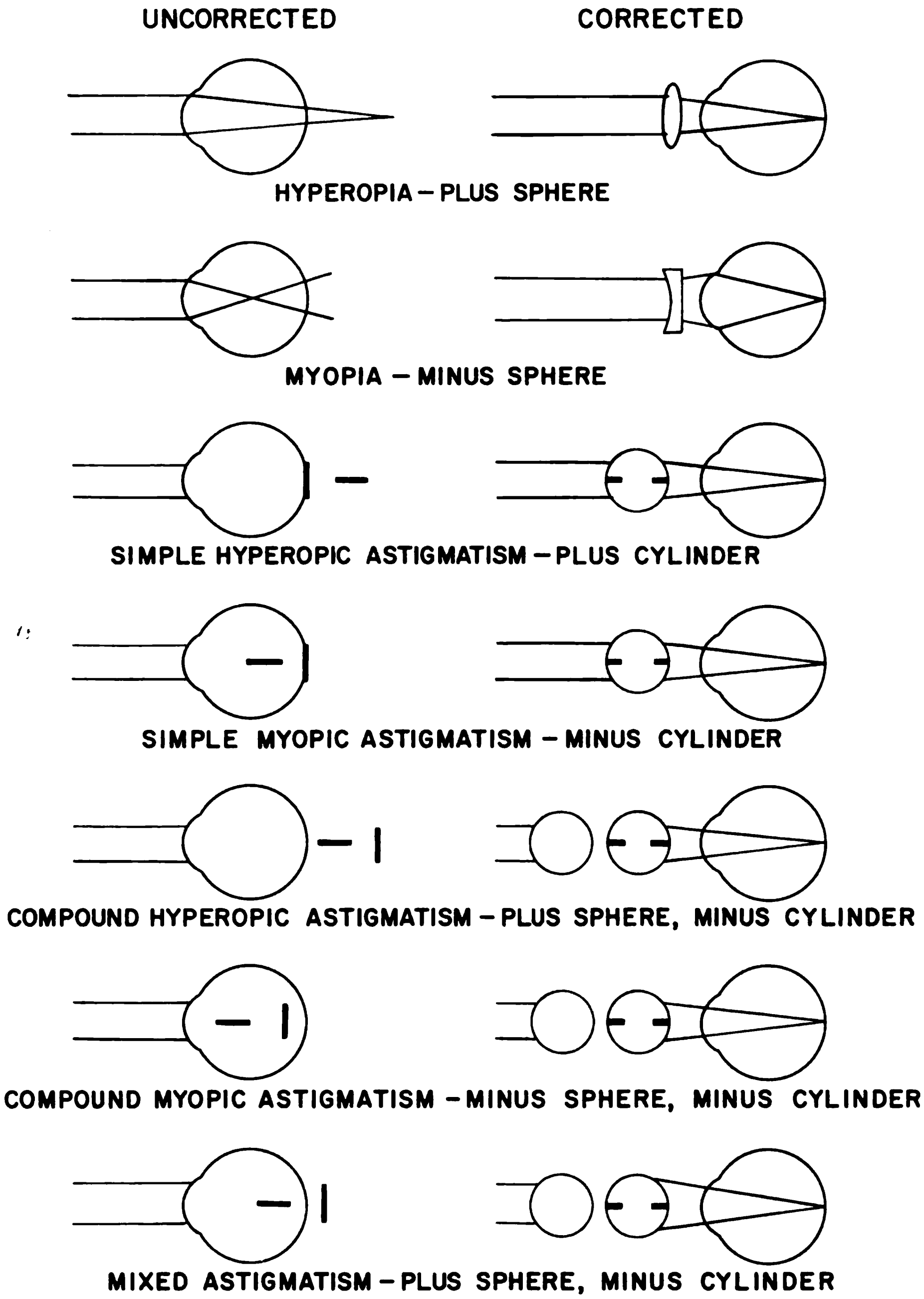
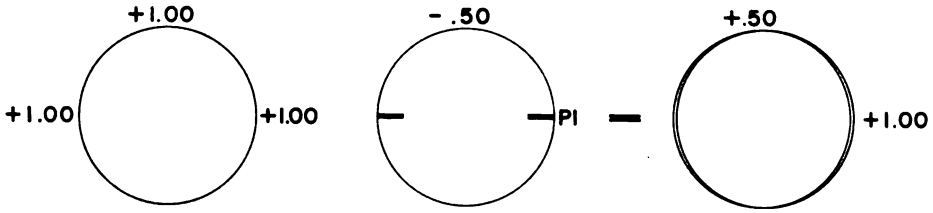

UNCORRECTED
CORRECTED
HYPEROPIA – PLUS SPHERE
MYOPIA – MINUS SPHERE
SIMPLE HYPEROPIC ASTIGMATISM – PLUS CYLINDER
SIMPLE MYOPIC ASTIGMATISM – MINUS CYLINDER
COMPOUND HYPEROPIC ASTIGMATISM – PLUS SPHERE, MINUS CYLINDER
COMPOUND MYOPIC ASTIGMATISM – MINUS SPHERE, MINUS CYLINDER
MIXED ASTIGMATISM – PLUS SPHERE, MINUS CYLINDER

FIGURE 9–15. *Types of lenses used for the correction of various refractive errors.*



+1.00 SPHERE -.50 CYL. AXIS 180

FIGURE 9–16. *Achieving desired lens power using a minus cylinder.*

it was written. It is necessary for the Flight Surgeon to know how to convert or "transpose" a prescription from minus cylinder form to plus cylinder form and vice versa. He might do a refraction (figure 9–18) on a patient using minus cylinders and the patient might be carrying a prescription by someone else written in plus cylinders. To determine if the two are the same, the following formula is useful:

1. Add algebraically the power of the present sphere and cylinder. This is the new sphere.
2. Change the sign of the cylinder (but use the same power).
3. Change the axis 90 degrees.

Still another way to obtain the power which was obtained in figures 9–16 and 9–17 is by using two cylinders. While this is no longer used in writing lens prescriptions, it is important in understanding refraction. The desired power can be obtained by taking a +1.00 cylinder and placing its axis at 90 degrees (power at 180) and adding to this a +.50 cylinder with its axis at 180 (power at 90).

REFRACTION

Refraction may be defined as any procedure followed to determine what lens power is needed to convert the patient to emmetropia or to give him good, comfortable vision. Refractive error is expressed as the needed corrective lens power. It is not the error itself. In other words, a —1.00 myope has one diopter too much refractive power. One might think his refractive error would be expressed as +1.00 but by custom it is not. It is —1.00, the power of the lens required to correct his vision.

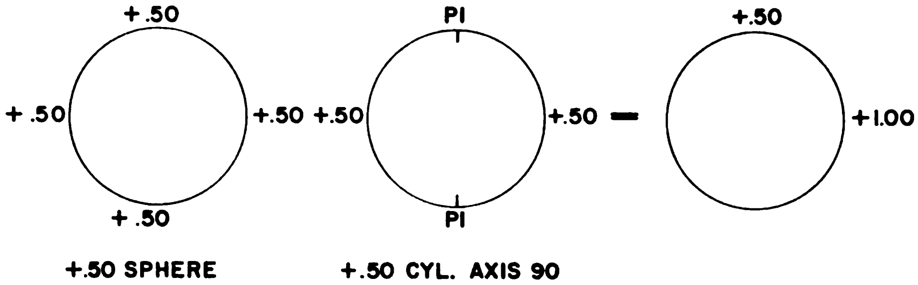


FIGURE 9–17. *Achieving the same lens power as in figure 9–16 using plus cylinder.*

Refraction can be categorized in several ways. For example, an administrative refraction is one performed on a candidate for flight training. It is unlikely that he needs glasses and the intention is not to prescribe but to determine what his basic refractive error is. A clinical refraction, on the other hand, is one done to determine if the patient needs glasses and, if so, of what power. Refraction is termed cycloplegic or noncycloplegic depending upon whether or not a cycloplegic agent is used to produce flaccid paralysis of the ciliary muscle. Administrative refractions are done under cycloplegia. Clinical refractions may or may not be. Refraction is further classified by method of examination as either objective or subjective. In an objective refraction, an objective measurement of the eye is taken using a retinoscope; in a subjective refraction, the patient's own responses are used in determining the lenses required. Refractionists (the term is inclusive, referring to both optometrists and ophthalmologists) differ in their opinions as to which method is the more reliable. The consensus is, however, that it is best to do both and try to achieve some correlation between the two. In examining any refraction patient or subject, part of the procedure may prove to be unsatisfactory and one must then rely upon the other methods. Many

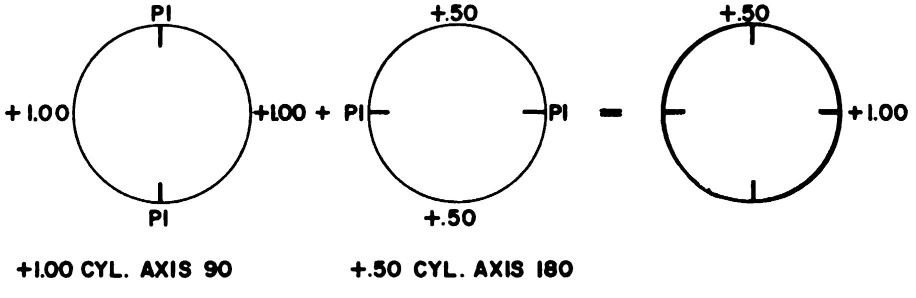


FIGURE 9–18. *Achieving the same lens power as in figures 9–16 and 9–17 using two cylinders.*

authorities feel that retinoscopy (objective) is preferable, particularly in preventing gross error, and that the subjective examination is more accurate for purposes of prescribing. This does not hold true for all individuals, of course.

The process of refraction is described below briefly. Discussion in depth, citing numerous examples of each situation which might be incurred, is not deemed necessary in this section. The qualified Flight Surgeon who has not had occasion to refract for some time may find this section useful for review. The novice to refraction will, of course, require the help of an experienced person in order to become qualified to perform actual refractions. It is hoped, however, that even the novice may achieve some understanding of the problems of refractive error whether he is able to put it into practice or not.

Retinoscopy

A retinoscope consists of a handle containing a light source and an eyepiece containing a mirror with a hole in it through which the retinoscopist looks. The mirror may be plane or concave. The widely used Copeland scope has a cuff on the handle. When the cuff is pushed all the way up toward the eyepiece, the result is that of a plane mirror. This is the recommended position with this scope since the retinoscopic figures and motions used in this chapter describe what is seen when using plane mirror scopes. If the cuff on the Copeland scope is allowed to slide down the handle, it has the effect of a concave mirror and everything will be reversed. Should the Flight Surgeon pick up a scope with which he is not familiar, he should shine the light on his hand while holding it close to the eyepiece, and then move his hand out from the scope. If the rays diverge all the way without crossing and then diverging, it is a plane mirror scope. In using the scope, the light is aimed directly into the patient's eye and moved up and down or side to side across his pupil. If the reflex observed moves the same way in his pupil as the light is moving, it is called a "with motion," and plus lenses should be added until the reflex fills the pupil and there is no apparent motion. This point is called the neutralization point. If the reflex in the patient's pupil moves opposite to the way the light is moving, this is called an "against motion," and minus lenses should be added until the neutralization point is reached. If the examiner were standing at 20 feet to perform the retinoscopy, the lens required to neutralize any meridian would represent the refractive error in that meridian. Since this is impractical, however, he subtracts (or adds minus algebraically) a diopteric power which is the inverse of his distance from the patient's eye. It is customary to assume that the "working distance" is one meter and --1.00 is added to the neutralization power to obtain the refractive error in that meridian. If the 90 degree meridian neutralizes at +1.00, —1.00 is added, which results in a power of plano. If it neutralizes at —1.00, —1.00 is again added, showing the refractive error to be —2.00 in that meridian. If the eye is "spherical," that is, if it is either hyperopic or myopic without astigmatism, all meridians will neutralize at the same point. If there is astigmatism, the two principal meridians must be neutralized (the meridians of greatest and least curvature or power). If the two principal meridians are not readily apparent, the vertical and the horizontal are neutralized. To avoid confusion, the neutralization points are noted on a cross as shown below. After the working distance has been subtracted, the refractive error is recorded on an angle. Should the 90 degree meridian neutralize at +1.50 and the 180 at +1.00, this is first recorded on the cross; then the working distance is subtracted to find the refractive error in each meridian. This is recorded on the angle as +.50 in the 90 and PL in the 180. This process is shown in figure 9–19. This must be converted to a prescription. Since, as a rule, only minus cylinders are used in performing refraction, the prescription should be written in that way. Therefore, the meridian of most plus is selected as the sphere, in this case +.50. The difference between the two is the cylinder. Here, it is the difference between +.50 and PL, which is —.50, and the axis is placed in the meridian of most plus, so that its action will be 90 degrees away. In this case, the most plus is in the 90 degree meridian and the action will be at 180, bringing that meridian back to PL. The prescription then is +.50—.50 x 90.

In summary, the steps to follow in retinoscopy are:

1. Locate the principal meridians. If they are not readily apparent, use the 90 and 180.

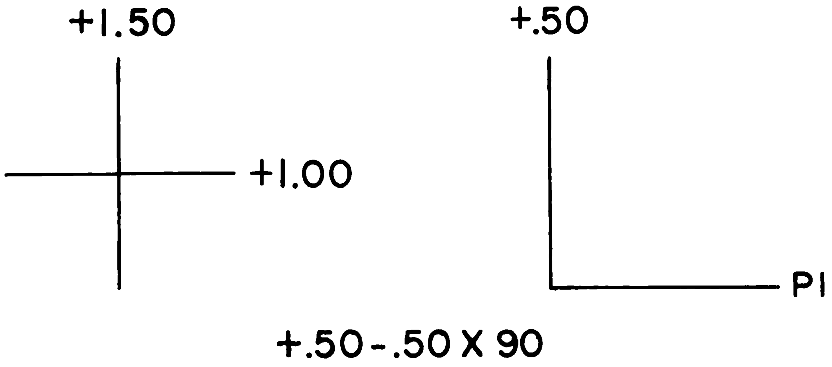


Figure 9–19. *Determination of prescription by means of retinoscopy.*

2. Neutralize the two meridians by adding plus spheres for a "with motion," minus spheres for an "against motion." Record on the cross.

3. Subtract an appropriate amount for working distance (if one meter, —1.00), and record on the angle.

4. Write the prescription by taking the meridian of most plus (or least minus) as the sphere, the difference between the two as the minus cylinder, and placing the axis in the meridian of most plus (or least minus).

Acceptance

Acceptance refers to how close the patient comes to accepting the retinoscopic findings, and how they can be refined to provide the best visual acuity. To accomplish this, the retinoscopic findings are placed before the patient's eye either in the trial frame or phoropter, to determine if he can read 20/20 or better. The sphere is then adjusted by a .25D up or down to get the best vision. The cylinder is then refined, using the Jackson cross cylinder. The cross cylinder has two red dots (or lines) which represent the axis of the minus cylinder; 90 degrees from these are two white dots (or lines) which represent the axis of the plus cylinder. It is important to remember that the action is 90 degrees from the axis in this as in all other cases. Cross cylinders can be obtained in powers of +.25, —.25; +.37, —.37; +.50, —.50; and +1.00, —1.00. Generally, the +.25, —.25 is the most useful. It can be thought of as a +.25 cylinder with its axis with the white dots combined with a —.25 cylinder with its axis with the red dots. A prescription for this would be +.25 —.50 x (the axis of the red dots). To refine the axis the handle of the cross cylinder is placed in line with the axis of the existing cylinder in the trial frame or phoropter so that the dots straddle that axis. Then it is flipped so that the red dots are first on one side of the existing axis and then the other (conversely with the white dots) and the patient is asked which seems clearer to him. The axis of the existing cylinder is moved toward the axis of the red dots in that position. This is repeated until the patient states that there is no difference or until he begins "bouncing" back and forth between two adjacent meridians. It is best to move the axis of the existing cylinder by 15 degree increments until there is a reversal of direction and then refine by 5 degree increments.

To refine the power, the dots (either white or red) are placed in line with the axis of the existing cylinder and, again, the cross cylinder is flipped.

This provides two positions, one in which the red dots are in line with the existing cylinder, and one in which the white dots are in that line. If the patient prefers the red dots, more minus cylinder is added. If he prefers the white dots, minus cylinder is taken out. What is being done with the cross cylinder in refining both the axis and the power, is expanding and contracting the little remaining Conoid of Sturm, and the patient will state that the eye chart is clearer in the position that gives the least Interval of Sturm and smallest Circle of Least Diffusion.

After refining the sphere and then the cylinder, a note is made of the patient's "acceptance."

Subjective Refraction

To perform subjective refraction, it is necessary to employ astigmatic dials of which there are many, many types. Among the most popular are the Lancaster-Regan Dials. Dial No. 1 has radiating lines every 10 degrees, while Dial No. 2 consists of two crossed lines at 90 degrees from each other mounted upon a revolving disc that can be rotated to any position.

In subjective refraction, the first step is to try to convert the patient to myopia, if he is spherical; or to compound myopic astigmatism, if he is astigmatic. This is done with lenses in the following manner. If he reads 20/20 unaided, he obviously cannot be very much myopic or astigmatic. Plus lenses are then added until his vision is blurred to (approximately) 20/40. This process moves the focal point or focal lines up into the vitreous and is called "fogging." If he is not reading 20/20, the nature of his error cannot be known unless an objective refraction has previously been done. A plus lens is tried. If he sees better, the examiner will conclude that he is hyperopic, and will continue adding plus lenses until the patient's best vision is attained—then still more until his vision is again blurred to about 20/40. If the original plus lens makes him worse, minus lenses (spheres) are added until the patient can read about 20/40. What is desired is to have the focal lines in the vitreous and yet close enough to the retina to allow for some degree of discrimination in that portion of the exam that follows.

The patient is asked to look at the center of L-R Dial No. 1 without squinting and to state if any lines appear to be clearer, blacker, and sharper than the rest. The refractionist should walk up to the chart and by pointing to the lines find out which line or lines are the blackest, etc. If there are several, the line in the center of the spread is picked. L-R Dial No. 2 is then set to match this line. Minus cylinders, with their axis 90 degrees from the clearest, blackest, sharpest line, are added until the two are equal. Each time .50D of minus cylinder is added, +.25 sphere is added (or —.25 sphere is taken out if patient is a myope). This causes the Interval of Sturm to collapse from both anterior and posterior toward the Circle of Least Diffusion. If the plus sphere is not added, it will collapse by moving the anterior focal line toward the posterior and there is danger of losing the "fog" and getting false answers from the patient. This is true, particularly, if there is a high degree of astigmatism, but it is well in any case to form the habit of adding .25D of plus sphere every time two quarters of minus cylinder are added. Once the two lines on L-R Dial No. 2 are equalized, it may be well to go one-quarter diopter beyond to make sure of getting a reversal and then to come back to the cylinder that equalizes them. All that then remains to be done is to reduce plus sphere, one-quarter diopter at a time or to add minus spheres in the same way until the patient reaches 20/20 or best vision. Using this subjective method has resulted in a greater percentage of satisfied refraction patients, providing a retinoscopy and acceptance have been done to avoid any gross error. Even for an administrative refraction, the subjective is a highly reliable method provided it is done under cycloplegia and is preceded by an objective refraction.

An explanation of what happens to the focal lines during subjective refraction and of why the subject sees certain lines on the astigmatic dials more clearly may be helpful to the understanding of this type of refraction. In the first step, the patient's vision is fogged to about 20/40. The reason for doing this is that, should the patient be an astigmat, both focal lines should be in the vitreous. The aim is to convert him to a compound myopic astigmat. In other words, and as mentioned earlier, the focal lines should be close enough to the retina to allow him some discrimination. In the case of a patient who has astigmatism with the rule, the vertical meridian of his cornea has the sharpest curvature and the horizontal meridian has the flattest curvature. This amounts to a spherocylindroid or toric lens. In the paragraph concerning lens power, it was shown that any toric lens can be thought of—in fact can be duplicated—by placing two cylinders together with their axes 90 degrees from each other. Therefore, the cornea of this patient with astigmatism with the rule should be visualized as consisting of two cylinders, the most powerful of which has its axis in the 180 meridian and its power, or curvature in the 90 degree meridian. The least powerful of these two cylinders has its axis in the 90 degree meridian and its power or curvature in the 180. Each of these two cylinders has a focal line just like any other cylinder and its focal line is parallel with its axis. The focal lines of these two cylinders which make up this cornea are in fact the two focal lines of the Conoid of Sturm produced by the astigmatic cornea, and it can be seen that the focal line controlled by the vertical curvature is horizontal and the focal line controlled by the horizontal curvature is vertical. Since the vertical curvature of the cornea in the present example of astigmatism with the rule is the strongest, the horizontal focal line which it controls will be anterior and, of course, the vertical focal line will be posterior. This is always true in astigmatism with the rule. In astigmatism against the rule the posterior focal line is horizontal and the anterior focal line is vertical. It can, in fact, be said that the posterior focal line in any case of astigmatism is always parallel with the strongest meridian of the cornea, yet it is controlled by the weakest meridian.

To return to the case in point—that of astigmatism with the rule which has been converted to compound myopic astigmatism with the rule by "fogging"—that is, if the patient was not already such to start with—when the patient is asked to look at the astigmatic dial, he will state that the vertical line is the clearest, sharpest, and blackest. He does this for the following reason: Since, it will be remembered, a conoid is a ray of light from a single point in space, and retinal images are made up of innumerable conoids, and since both focal lines are in the vitreous, the image on the patient's retina is being made up of the blurs posterior to the posterior focal lines of these innumerable conoids. The conoid posterior to the posterior focal line is elongated or blurred vertically. If the patient looks at the lines on the astigmatic dial and if the image of each point on each line is blurred vertically, the vertical line will appear thinnest and sharpest and blackest, and the horizontal line will appear thicker and grayer. So it can be said that in astigmatism with the rule, if the patient's vision is properly fogged, he will always see the vertical focal line as clearest, sharpest, and blackest. The appearance of the astigmatic dial is shown in figure 9–20. In astigmatism against the rule, the patient will see the horizontal line as clearest, sharpest, and blackest; in fact, wherever the axis of the astigmatism is, the line that is clearest to the patient will match the meridian of sharpest curvature of his cornea.

After it has been determined which line on L–R Dial No. 1 is clearest, L–R Dial No. 2 is set to match. This merely delineates the problem for the

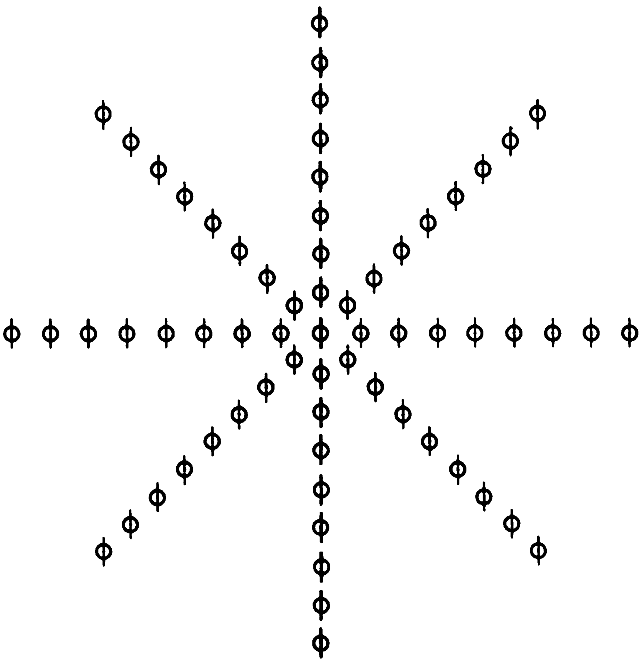


FIGURE 9–20. *Appearance of astigmatic dial in a case of astigmatism with the rule. Vertical line is blackest because the image of each point on that line is blurred vertically. Some lines have been eliminated for simplification.*

patient. Minus cylinders are then inserted 90 degrees from the clearest and blackest line. In the case of the patient with the sharpest curvature in the 90 degree meridian, the minus cylinders are placed so their axes are in the 180 degree meridian. This weakens the power of the whole system in the vertical meridian. The vertical meridian controls the horizontal focal line, which in this case is anterior, and the minus cylinder which is added forces the anterior focal line back. More minus cylinders are added, .25 diopters at a time, until the two lines on Dial No. 2 are equalized. Each time two increments of minus cylinder are added, .25 diopter plus sphere is added, which causes the Interval of Sturm to collapse from both ends toward the Circle of Least Diffusion. When the two lines on Dial No. 2 are equal, the Interval of Sturm has been collapsed and the patient's vision is now "spherical." All that remains to be done is to reduce the plus sphere (or increase the minus sphere) so that the new point focus is pushed back to the retina. It can be assumed this has occurred when the patient reads 20/20 or better. It is important to know that each time .25D of plus sphere is removed or .25D of minus sphere is added, it should improve the patient's vision on the Snellen Eye Chart by about one line.

Accommodation has been previously defined as the ability of the eye to increase its refractive power by action of the ciliary muscle on the lens causing it to assume a more globular shape. Everyone loses accommodative power as he grows older at a fairly standard rate. This loss is due both to the loss of power of the ciliary muscle and to the fact that the lens becomes harder and less malleable as age increases. The process is uniform in all people. By calculating an individual's true accommodative power, that is, by first determining his refractive error and then seeing how much he can accommodate with the correction in place, his age can quite accurately be guessed. Age 42 is said to be the "bifocal age" or the age at which one can no longer achieve good, clear, distant vision and good, clear, near vision with the same pair of glasses unless they are bifocal. This is called presbyopia. Obviously, not every 42-year-old wears bifocals. The 42-year-old emmetrope sees well at distance, and early in the day he can also read well. In the evening, however, his near vision begins to blur a little. If a 42-year-old has a small degree of myopia, his vision will be a little blurred at distance, but he can see well nearby until he is a few years older, perhaps 45. If a 42-year-old is a hyperope, he will need full hyperopic correction a little before 42, and when he is 42, he will need something additional for his near work. A myope of sufficient degree to necessitate his wearing glasses all, or most, of the time will require bifocals at about age 42, because with his glasses on he is a false emmetrope, and therefore subject to the same phenomenon with the exception that he can if he wishes take off his glasses to read.

It takes +3.00 diopters of accommodation for anyone to shift his gaze and focus from a distant object to a near one at reading distance. Reading distance for the purpose of calculation is assumed to be one-third of a meter. Light rays reaching the eye from a distance of one-third of a meter have a negative vergence of —3D. Therefore, the eye must generate +3D to overcome this and place the image on the retina. An emmetrope would accommodate only +3D, but a +2D hyperope would have to accommodate 2 diopters for distance and 3 more for near—or a total of 5D. A —2D myope without his glasses, on the other hand, is already in focus at 50 cm and has to accommodate only one diopter to see at 33 cm.

It is obvious then that accommodation and presbyopia constitute a problem with the older pilot. If he cannot accommodate enough to read his instruments and his radio gear, he must be furnished suitable glasses to enable him to do so. The accommodative power of the young applicant for flight training is also of interest. An occasional individual has premature presbyopia, although such cases are rare. The Flight Surgeon would not want such an individual to slip by into the flight program. Occasionally, a case of hyperopia of significant degree may remain undetected, either through error in refracting or failure to obtain full cycloplegia. These cases can be detected by measuring the accommodation with the Prince Rule. The Prince Rule is a rule 0.5 meters long with a slide which holds a card on which there is fine print. On the back of the rule there is a scale which shows age in one column and the standard accommodation for that age in another. Each eye is tested separately. The end of the rule is placed against the base of the bridge of the nose with the reading card close to the subject's eye. The card is moved slowly out away from the eye and the subject is asked to state when the print comes into focus. He is then asked to read the letters. The "accommodation" is then read off the scale on the back of the rule. For applicants for flight training it must be within 3 diopters of the standard for age. Older aviators must have a minimum of 2.5D of accommodation or be corrected to that amount. "Accommodation" is written in quotes above because the Prince Rule does not actually measure accommodation. It measures accommodation plus or minus refractive error. However, it is a simple test and it serves the purpose very well. If a candidate scores accommodation considerably better than the standard for age, he may be a myope who has somehow "got by" the examination so far. If he scores more than 3 diopters below the standard for his age, he is probably more hyperopic than the +2.50 which is allowed, or more astigmatic than the 0.75D cylinder that is allowed.

Bifocal Refraction

Bifocal refraction must always be prefaced by a distance refraction. The tendency of the novice refractionist is to say to himself, "I have here a man who reads 20/20 O.U. He doesn't want anything for distance. Why don't I just throw some plus spheres up before him until I get him to reading well?" There are a number of reasons for not doing so. First of all, it is like picking out glasses at the dime store, albeit this system sometimes works. The patient may be emmetropic in one eye and +1.50 in the other. If he gets the same amount of correction in each eye, one eye will have to work harder than the other. He may have a significant amount of astigmatism, say 0.75D, and still read 20/20. If spheres are prescribed, he will not have the comfort or the clarity he would have if the proper cylinder were included. First, therefore, a normal distance refraction is done. There are any number of ways to determine the "Add" or his bifocal segment. Only one will be described. A standard add for age (see below) is inserted before each eye, and a check is made to see if he can read the smallest print on the reading card. The card is moved closer to each eye, separately, to see where it blurs. It should blur at the same distance in each eye. If it does not, the distance correction is checked, and the examiner should be alerted to the possibility of some eye pathology. Only as a last resort are different adds given in the two eyes. If the two eyes do blur at about the same distance, it is an indication that the correction is well "balanced" for the two eyes. Next, the near prescription is set up in the trial frame, and the patient is given the reading card and asked to move it in and out until he gets his best focus, using both eyes. This should be at about 17 inches. If closer, the add is reduced; if further, the add is increased. The patient is asked to bring it up closer to his eyes; it should blur at about 10 inches. When he moves it farther away from his eyes it should blur at about 23 inches. The stronger the add, the smaller is the depth of field, so in strong adds, the near blur may be at 12 inches and the far blur at 20 inches. The point of best focus should nearly always be at 17 inches, although this may vary for occupational reasons or if the patient has formed the habit of holding his reading matter closer or further than this.

Standard Adds for Age

42	+0.75 to +1.25
45	+1.50
48	+1.75
52	+2.00
55	+2.25
58	+2.50

No more than +2.50 add is ordinarily given unless the patient has had a lens extraction for cataract in which case +3.00 is given.

Trifocals

Many people wear trifocals who do not actually need them but nevertheless seem to enjoy them. They are intended for special occupational or avocational use. One such use could be for the older aviator who cannot see his instrument panel clearly through his distant correction and cannot see it with his bifocal segment unless he leans way over and holds his chin in the air. In such a case, a trifocal would be very helpful. The add in the intermediate segment is always half the add in the lower segment (the add in the lower segment is the same as the add would be in a bifocal). This works out fortuitously because the lower segment can be used for near vision, such as reading maps and instructions. At the distance out from the eye that this becomes blurred, the intermediate segment becomes clear and can be used for reading the instrument panel.

Double bifocals, called "baseball bifocals" because of the line above and below like a baseball, are also useful to the older pilot who may be flying a multiengine aircraft with the radio gear mounted over his head. Putting an add in a segment in the top part of his glasses enables him to see this gear without having to contort himself in an effort to get it within view of his standard bifocal. This type has many other occupational uses, for example, for the man working in a parts room where there are parts in bins reaching from floor to ceiling, etc.

MUSCLE BALANCE OR OCULAR MOTILITY

Nomenclature of Muscle Balance

Just as there are few emmetropes, there are few people who do not have some tendency, at least, for their eyes to deviate either in, out, up, or down from the normal conjugate position of gaze. Those who do not have such tendency however are called *orthophoric* (meaning straight eyes). If there is a tendency for the eyes to deviate, yet they do not do so because of *fusion,* the individual is said to be *heterophoric,* and the condition is known as *heterophoria.* An actual deviation of the eyes is called *heterotropia* and the person is said to be *heterotropic.* Fusion is the ability of the two eyes to "lock on" to the same object or objects of regard and to function together producing one mental image.

Heterophoria is further subdivided as follows:

Esophoria—A tendency of the eyes to turn in.

Exophoria—A tendency of the eyes to turn out.

Hyperphoria—A tendency for one eye to turn up or the other to turn down. It is named either right or left by the "high" eye. If there is a tendency of the right eye to turn up, or of the left eye to turn down, it is called a right hyperphoria. The right eye tends to be higher in either case. Conversely, a tendency for the left eye to turn up or the right eye to turn down is a left hyperphoria. (It should be noted that there is no term " hypophoria.")

Cyclophoria—A tendency for an eye to rotate around the anterior-posterior axis. If there is a tendency for the 12 o'clock meridian to turn toward the nose, it is an incyclophoria; if there is a tendency for the 12 o'clock meridian to turn away from the nose, it is an excyclophoria. Heterotropia is similarly subdivided:

Esotropia—One eye turns in, or they alternate. Crosseyed.

Exotropia—One eye turns out, or they alternate. Walleyed.

Hypertropia—If the left eye is looking at the object of regard and the right eye is looking up, this is a right hypertropia. Also, if the right eye is looking at the object of regard and the left eye is looking down, it is a right hypertropia. Conversely, if the left eye is looking at the object of regard and the right eye is looking down, or if the right eye is looking at the object and the left eye is looking up, it is a left hypertropia.

Cyclotropia—It is incyclotropia if the 12 o'clock meridian is turned toward the nose; excyclotropia if it is turned away from the nose.

All tropias of whatever degree are disqualifying for flight training or for continuation as an aviator if they develop or are discovered after designation for the reason that, in a tropia, fusion has been broken and there can be no stereopsis. Further, the individual who has a tropia must suppress the vision in one eye or he will have diplopia. Although many private pilots do not have fusion and there have even been a number of one-eyed pilots, it is generally agreed that military pilots should have good fusion and stereopsis.

In the case of phorias, there are certain allowable limits as to precisely how much of a phoria there can be. The reason for having such standards is that high degrees of phoria tend to break into tropias under conditions of fatigue. If a pilot were allowed to fly with more of a phoria than that which is considered normal or acceptable, he might return from a lengthy and tiring flight and find himself experiencing diplopia, just as he was about to make his approach for a landing.

Measuring Ocular Motility

Since there are standards, there must also be methods for measuring the degree of ocular muscle imbalance. There are a number of ways of doing so.

The Hirschberg Examination.—The Hirschberg examination is the simplest of the tests, and to perform it one shines a small pen light (called a "muscle light" in ophthalmology) toward the bridge of the patient's nose, asking the patient to look at it. If the eyes are in alinement, the reflection of the light will be located at the same spot in both of the patient's pupils, usually just a tiny bit nasal to center. If one eye is turned in, the reflex in that eye will be located temporal to center, and if one eye is turned out, it will be located nasal to center. The degree of turn can then be estimated as follows: halfway between center of pupil and the edge of an average pupil, 10°; over the edge of an average pupil, 25°; halfway from pupil edge to limbus, 35°; over the limbus, 45°; and out on the sclera, 60° (figure 9–21).

The Krimsky Examination.—This is actually a modification of the Hirschberg in which prisms are introduced in front of the deviating eye and increased until the reflex appears centered in each eye. The amount of turn is then measured in prism diopters. One prism diopter will divert a ray of light one cm in a distance of one m. The deflection is toward the base of the prism. A 2-prism-diopter prism will divert a ray of light 2 cm in a distance of one m. In the Krimsky and in all other methods in which prisms are used to measure phorias or tropias, the prism must always be pointed in the direction of the turn. To measure an esotropia by the Krimsky method, a base-out, point-in prism is used. Both the Hirschberg and the Krimsky are useful only in measuring tropias, and while they do not appear to have much application to aerospace medicine, they do contribute to an under-

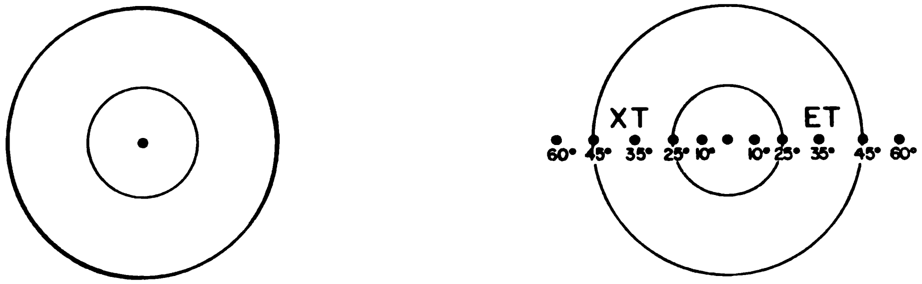


FIGURE 9–21. *The Hirschberg Examination.*

standing of the subject of motility, and they may be useful to the Flight Surgeon should he be called upon to examine nonaviation personnel.

The Maddox Rod.—The Maddox Rod is used to break up fusion. It is a lens made up of a series of small cylinders of about 80 diopters power. A ray of light passing perpendicularly through the lens is brought to a focus in a series of small dots one-eightieth of a meter behind the lens, and the dots are elongated perpendicularly to the axis of the cylinders or "rods." If one looks at a light source with one eye bare and the other covered with the Maddox Rod, the uncovered eye sees the light and the other eye sees a line of light. The brain cannot fuse such dissimilar images and fusion is thus broken. Some Maddox Rods are made of red glass so that one eye sees a white light and the other sees a red line, thus creating even further dissimilarity. When fusion is broken, the eyes assume a relaxed position or their position of muscular imbalance. In observing what happens with various types of phorias, the Maddox Rod can be placed before either eye and the same results obtained but, for the sake of consistency, it is placed before the right eye in each of the following cases.

1. *Esophoria.* In testing for horizontal phorias a vertical line is used; therefore the rods are placed horizontally (see figure 9–22). The left eye is looking at the light, fusion has been broken, and the right eye is now deviating in. Light from the target passes through the Maddox Rod, assumes the form of a line, and strikes the nasal retina. Anything perceived by the nasal retina is interpreted as coming from the temporal field. The patient

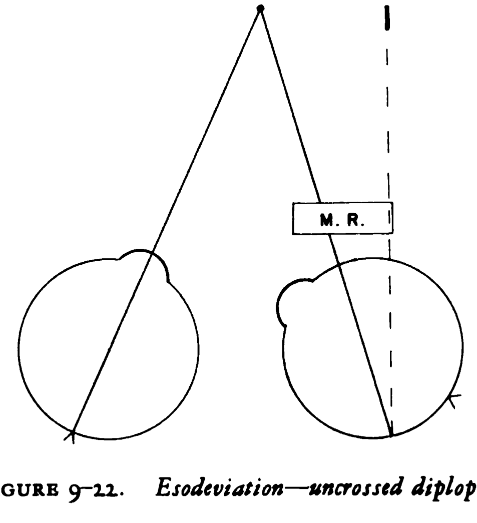


FIGURE 9–22. *Esodeviation—uncrossed diplopia.*

therefore sees the line to the right of the light. This is called "uncrossed diplopia," and it is always to be found in esodeviations. It is called uncrossed because the rod is over the right eye and the image it produces is to the right.

2. *Exophoria.* The left eye is again looking at the light (see figure 9–23). Fusion is broken by the Maddox Rod. The right eye now turns out. Light coming from the target passes through the rods, is changed into a line, and the image is perceived by the temporal retina. Anything seen by the temporal retina is interpreted as coming from the nasal field, so the patient sees the line to the left of the light. This is called "crossed diplopia," and it is characteristic of exodeviations. Crossed diplopia refers to the fact that though the Maddox Rod is over the right eye, the line which it produces is seen to the left. It is also remembered in this way—by thinking of the X in eXo, and the fact that an X is also a cross.

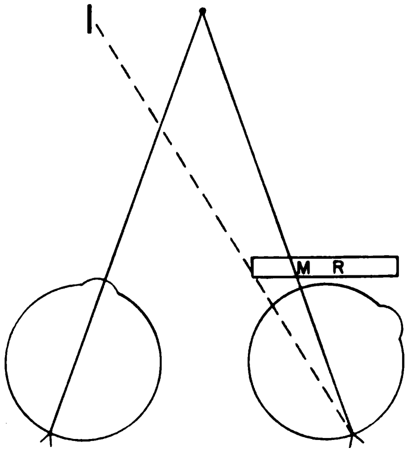


FIGURE 9–23. *Exodeviations.*

3. *Right Hyperphoria.* Here again, the left eye is looking at the light. (see figure 9–24). Fusion has now been broken and the right eye is looking up. Light from the target passes through the Maddox Rod, is changed into a line, and the image falls on the superior retina. (Horizontal lines are used for vertical deviations.) Anything seen by the superior retina is interpreted as coming from the inferior field, so the line is seen below the light. It helps to remember, the *low image is the high eye.* Since the line is seen below the light, this is a right hyperphoria.

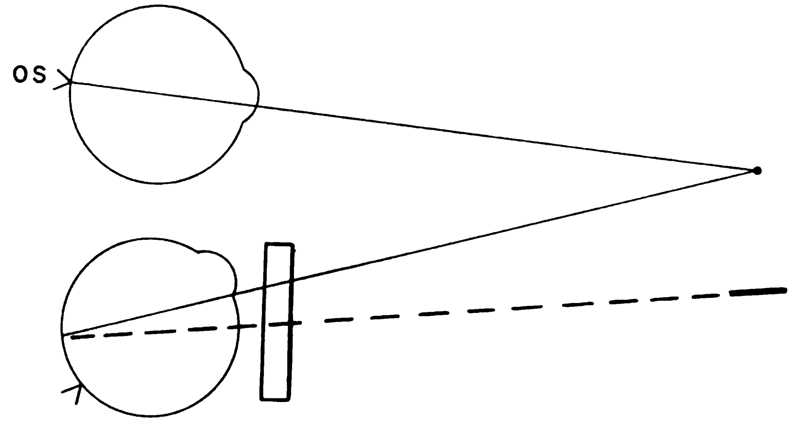


FIGURE 9-24. *Right hyperphoria.*

4. *Left Hyperphoria.* The left eye is looking at the target light (see figure 9-25). Fusion has been broken, and the right eye is looking down. Light from the target passes through the Maddox Rod, is changed into a line, and the image falls on the inferior retina. Anything seen by the inferior retina is interpreted as coming from the superior field, so the subject sees the line above the light. Again, "the low image is the high eye." In this case the image of the light is the low image and it is perceived by the left eye. This is a left hyperphoria.

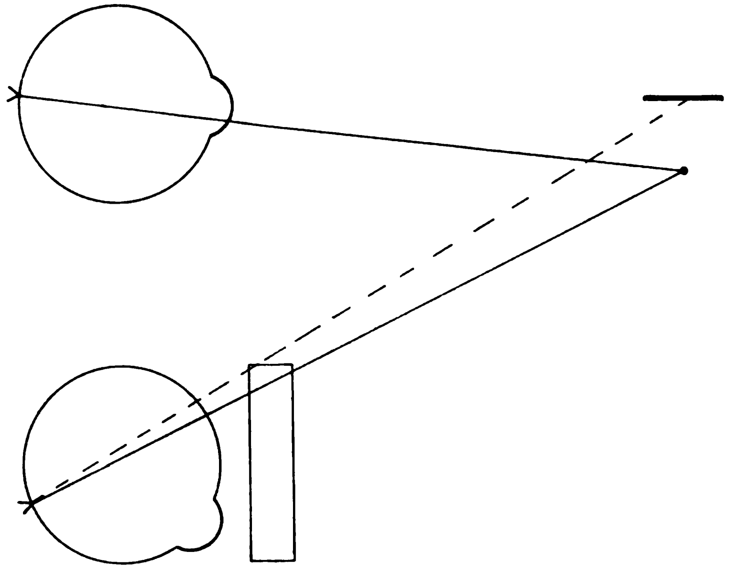
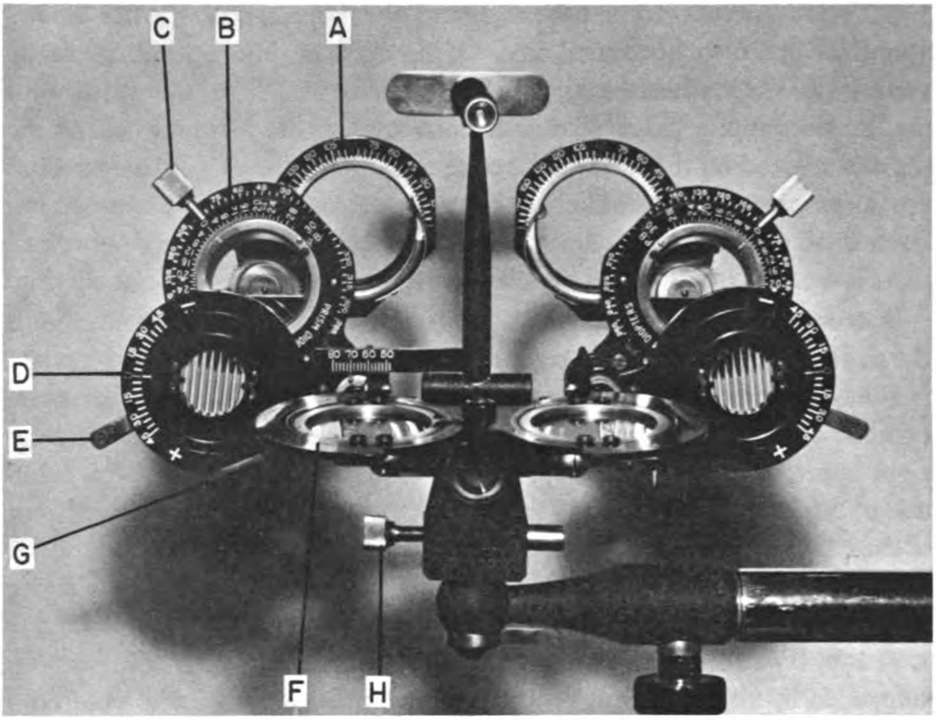


FIGURE 9-25. *Left hyperphoria.*

5. *Cyclodeviations.* The patient should always be asked whether he sees a vertical line or a horizontal line. If he says the line is neither exactly vertical nor exactly horizontal, then the Maddox Rod should be tilted until the line does appear either vertical or horizontal, as the case may be, and the degrees are read off the scale on the edge of the instrument. Cyclodeviations are uncommon and are unlikely to be encountered in aviation examinations. Even small degrees of cyclodeviation are difficult to overcome by fusion, and there is usually an associated vertical deviation sufficient to be disqualifying.

In order to measure the deviation, a prism is introduced in each case until the patient says the line passes right through the light. To do this, the prism is always pointed in the direction of turn. For an esophoria, a base-out, point-in prism is used, for an exo, a base-in, point-out prism is used; for a right hyper, base-down, point-up, in front of the right eye—or base-up, point-down, before the left eye; and for a left hyper, base-down, point-up, in front of the left eye—or base-up, point-down, before the right. This could be done with loose prisms but it is much faster and more accurate with the Risley Rotary Prism (see figure 9–26).

The Risley Prism consists of two 15-prism-diopter counterrotating prisms controlled by a thumbscrew. When the two prisms are point to point and base to base, they exert 30 prism diopters of power. When they are arranged base to point, they exert no power, and at points in between they exert partial power which can be read off the scale on the periphery of the assembly. To measure horizontal deviations the thumbscrew should be vertical, and of course the Maddox Rods should be horizontal in order to produce a vertical line. When the thumbscrew is turned clockwise, base-out prism is introduced and the amount can be read off the scale. This would be used if the patient gave an "uncrossed" response when presented with the Maddox Rod. When the thumbscrew is turned counterclockwise, the base moves inward and this would be used when the patient gave a "crossed diplopia" response. To measure vertical deviations, the thumbscrew is placed horizontally and the Maddox Rods are placed vertically so that there is a horizontal line. Base-up or base-down prism can then be introduced as appropriate. The Risley Prism, however, is graduated in diopters and for the purposes of aviation examinations, something more accurate is needed when it comes to the vertical phorias. Vertical phorias are more difficult to tolerate and maintain fusion; therefore the standards for vertical phorias are much more strict than for horizontal phorias (i.e., 10 prism diopters of exophoria or 5 prism diopters of esophoria are allowed, while only one prism diopter of



A—Trial frame
B—Risley prisms on zero position
C—Thumb screw for adjusting Risley prisms
D—Multiple Maddox rod
E—Adjustment lever for Maddox rod
F—Stephens prism
G—Adjustment lever for Stephens prism
H—Adjustment screw to Level Phorometer

FIGURE 9–26. *Phorometer.*

hyperphoria is permissible, with up to 1.6 under certain conditions). To measure vertical phorias, the Stephens Prism is used. It consists of prisms before each eye that are also counterrotating, but they are graduated in tenths of diopters.

There are several limitations to the Maddox Rod test: (1) The Maddox Rod does not always distinguish between a phoria and a tropia. It measures the imbalance, but only in certain instances does it give the examiner an idea whether there is a phoria or a tropia. While the Maddox Rod breaks up fusion, should the subject not be fusing in the first place, considerable ambiguity could occur. If the examiner did not see an obvious tropia or did not perform some other test, he would not know what he was measuring. (2) If the subject were an alternator, he could not see the line and the light at the same time, but would see first one and then the other. Although this would provide the examiner with the valuable information that the subject was, in fact, an alternator, it would be impossible to get a measurement in such a case. (3) Should the subject constantly suppress one eye, he would be able to see only one of the images. Again, this is important information but the angle of turn could not be measured. (4) If the subject were one who had had constant diplopia all his life, with which he had learned to cope, he would see both the line and the light at the same time but would not be able to tell the relative position of the two in space. When asked to state which side of the light the line was on, he would reply almost in consternation at the examiner's stupidity, "Well! I see the line with my right eye and the light with my left" (or vice versa, depending on which eye was looking through the Maddox Rod).

The Cover Test.—Although the Maddox Rod is the standard instrument for measuring phorias for aviation physicals, it is imperative that the Flight Surgeon also perform a cover test to rule out a tropia. The cover test is also useful in ruling out "Instrument Convergence." In certain individuals, convergence is stimulated whenever any large object such as the phoropter or phorometer is placed close to their faces, with the result that falsely high readings of esophoria are obtained. A simple cover test does not cause this convergent effect to such a great degree, and in these instances, it can be more accurate than the Maddox Rod test. Cover test findings can be used on the SF 88 (Report of Physical Examination), instead of the Maddox Rod findings if the examiner so desires. The cover test consists of three parts, (1) cover-uncover, (2) cross cover, and (3) prism and cover.

1. *Cover-Uncover Test.* This test can be performed at both near and far. The subject is asked to look at a target light. The examiner covers the subject's right eye (with his fingers or with a card or "paddle") and observes the subject's left eye. If it does not move, obviously the left eye is looking at the target as desired. The right eye is then uncovered, both eyes are given an opportunity to look at the target light, and then the subject's left eye is covered. If the right eye does not move, it is also looking at the target. If both eyes were looking at the target, there can be no tropia, and the subject is either orthophoric or heterophoric. When one eye is covered, should the other eye move outward and pick up fixation on the target light, this would show that prior to the covering of the fellow eye, this eye had not been looking at the target light at all but rather had been looking inward. This patient would, then, have an esotropia. Likewise, if the examiner covered one eye and the other eye moved inward and picked up fixation on

the target light, it would mean that that eye had been looking outward and the subject had an exotropia. If when one eye is covered, the other eye moves downward and looks at the light, then that eye had been looking up, and there is a hypertropia on that side. And finally, if when one eye is covered, the other eye moves upward and looks at the light, then it had been looking down, and the patient has a hypertropia on the opposite side. (There is, as mentioned earlier, no "hypotropia.") The cover-uncover test will reveal a tropia and will tell the examiner what kind of tropia it is. It does not rule out a phoria.

2. *The Cross Cover Test.* If a tropia has been ruled out by the cover-uncover test, a cross cover test is performed to look for a phoria. The patient looks at a target light. One eye is covered and instead of simply uncovering it as in the first part of the test, the cover is moved over to the opposite eye, and the examiner watches what happens to the eye that had previously been covered. If it moves outward, then it had been turned inward while covered and there is an esophoria (covering the eye serves to break up fusion). The cover is moved back and forth from right eye to left eye, and if each eye moves inward as it is uncovered, then it had been looking outward and there is an exophoria. If the right eye moves down as it is uncovered and the left eye moves up as it is uncovered, then there is a right hyperphoria. And if the right eye moves up as it is uncovered and the left eye moves down as it is uncovered, there is a left hyperphoria.

3. *Prism and Cover.* Having determined whether there is a phoria or a tropia by the first two parts of the test, the examiner proceeds to determine the degree by using prism and cover. To do this, he simply introduces prisms of increasing power while continuing to do cross cover, until he gets no further "jump" of either eye as it is uncovered and records the amount of prism that this requires. Here, as in all other methods of measuring phorias and tropias, the point of prism is placed in the direction of turn. One may wish to determine not only what prism it takes to neutralize the "jump" but what it takes to overcorrect it. As an example, a case may be imagined concerning a candidate who has been measured as having 8 diopters of esodeviation on the Maddox Rod. Over 5 is disqualifying, and the examiner wishes to determine whether or not the candidate is having "instrument convergence." Cover-uncover shows that he does not have tropia. Cross cover demonstrates the esophoria, and on prism and cover no further "jump" is observed with 2 prism diopters, or with 4 and 5 prism diopters, but at 6 prism diopters of point-in, base-out prism, the patient shows an

"Exojump." He is, therefore, overcorrected and the examiner knows that he cannot possibly have more than 5 prism diopters of esophoria. Prism and cover can also be done subjectively, by asking the subject which way the light seems to move during cross cover. The examiner introduces the proper prism of increasing size and finds out when the light appears to stand still during cross cover and when it begins to move in the opposite fashion.

Prism Divergence

Prism divergence is tested at both distance and near. It tests how much the eyes are able to diverge against the pull of a base-in prism and still maintain fusion. The subject looks at the target light and is told to maintain one image as long as he can and to mark the point at which it splits into two lights. The Risley Prism is used, and base-in prism is slowly introduced until diplopia is produced. The base-in prism causes the image of the light to be deflected toward the nasal retina and the eye deviates outward as long as it can in order to keep the image of the light on the macula (see figure 9–27). When it can no longer do so, fusion is broken and the eye quickly turns back inward to its position of rest, with the result that when the patient notes two images he also notes that they fly far apart suddenly. The motility standards state that the prism divergence for distance must equal the esophoria, if any. In other words, if the individual has any tendency for his eyes to turn in, the Flight Surgeon will want to know that he has an equal ability forcibly to turn them out under the

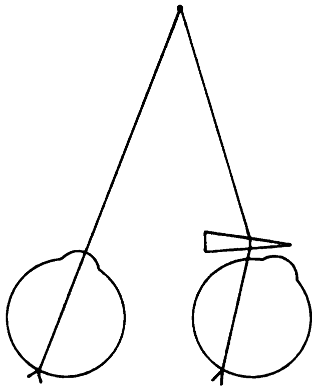


FIGURE 9–27. *Prism divergence—right eye diverging to keep image of light on macula.*

influence of the prism. This reduces the chances of his developing diplopia due to his esodeviation when fatigued. The standards also state that all candidates must have 12 prism diopters of prism divergence for near.

Near Point of Convergence

Instead of testing the prism convergence (which, incidentally, can be done in similar fashion to prism divergence by simply introducing base-out, point-in prism), the examiner may utilize the near point of convergence. On the SF 88, this is listed as P.C., although it may also be referred to as the N.P.C. To test the P.C., a meter stick or even the Prince Rule is placed alongside the bridge of the patient's nose. He is asked to look at a target light which is then moved in closer and closer to his nose while the examiner observes his eyes. When fusion breaks, one eye will suddenly move temporally while the other continues to follow the target. This point is noted in millimeters from the eyes and is recorded as the P.C. The standard for the P.C. is that it must be equal to or less than the P.D. or pupillary distance. More errors are made in trying to find the P.C. when it is remote than when it is normal or nearly normal. If it is remote, one may already be inside the P.C. at the start of the test. Then not seeing any "break" as the light is moved inward, one tends to record the P.C. as the point at which he is certain that the eyes are not in alignment. If the examiner is not certain, he should do a cover test to see if the subject is fusing at any distance. Some subjects do not converge well on a target light. In such a case they should be shown some fine print, asked to focus on some short word and look at it as the card is moved in toward the face. The act of accommodation increases the convergence power and gives a truer reading under these circumstances.

Fusional Tests

The Red Lens test is one test for fusion. For aviation purposes it is performed with the subject seated 75 centimeters from a wall or screen. A red glass is placed over one eye and the patient is asked to look at a target light held against the screen, or wall, in the primary position (i.e., directly in front, on a horizontal line). He is asked how many lights he sees and what color. Normally, he should see one pink or reddish light. Each eye is then covered consecutively to show him what the light would look like if he suppressed one eye. A prism can be held in front of one eye to produce diplopia and demonstrate it to the subject. Then the patient is instructed not to move his head (a chin rest is better) and to follow the light with his eyes. He is to tell the examiner if he sees two lights at any time and also if it changes color. The light is moved into the other cardinal positions: up and right, straight up, up and left, straight left, left and down, straight down, down and right, and straight right. There should be no diplopia within a radius of 50 centimeters from the primary position when the test is performed at 75 centimeters. It can be normal to have diplopia on extremes of gaze.

The Worth Four-Dot test, another fusional test, can be performed at both distance and near. For near testing, an ordinary flashlight is used. The light is shielded so that it shows a small white dot, two green dots, and one red dot. The subject wears a red glass over one eye and a green glass over the other. The eye with the red glass can see the red dot (this is red light, not a printed red dot) and it can see the white dot which it sees as red. The eye with the green glass sees the two green dots and the white dot which it sees as green. If the subject is fusing, his total perception will be four dots, either two reds and two greens or one red and three greens. If he is suppressing one eye, he will see either two red dots or three green dots; and if he is having diplopia, he will see three greens and two reds. The latter is not fully reliable, however, since the introduction of the red-green glasses themselves will tend to break up fusion. The Worth Four-Dot is not routinely used in aviation but nevertheless it is a good test to use in evaluating questionable cases.

The Keystone Stereopticon is a regular stereopticon with a set of stereo cards. One of the most useful cards has six rows of symbols. Two halves of the card are slightly dissimilar so that stereopsis is possible, and on each row of symbols one symbol should stand out from the card and appear closer to the observer. On each succeeding row, the dissimilarity of the cards is less (the angle of stereopsis is smaller) so that each one is a little more difficult than the line above. The card is graded in percent of stereopsis. This test is not a standard aviation test but it is useful sometimes in evaluating candidates who are having difficulty with the Verhoeff test. The Keystone is a very easy stereoscopic test. If a subject cannot pass all six lines on this test, it is unlikely that he will be able to pass the Verhoeff even with rest and repeated retesting.

The Verhoeff Test is the test used for testing depth perception. Actually it does not test depth perception, as is shown in the discussion of that subject. It tests only stereopsis, and if there is stereopsis, there is fusion in the highest degree.

Classifications of Horizontal Muscle Balance

As mentioned earlier, muscle balance can be measured both at distance (20′) and near (13″). Often the findings are quite different when this is done. Cases of horizontal muscle imbalance are classified according to where the deviation is greatest, provided there is a significant difference. In cases of esodeviations, whether phoria or tropia, if the deviation is greater near, it is called a convergence excess, and if the deviation is greater at distance, it is called a divergence insufficiency. In exodeviations, if the deviation is greater near, it is called a convergence insufficiency, and if greater at distance it is called a divergence excess. The difference should be at least 5 diopters to have any significance at all, and in some cases it is much greater. In the aviation physical examination, the phorias at near are not routinely tested. However, near point of convergence and prism divergence at near (as well as at distance) are tested. If either of these tests does not meet the standards, then the phorias should be tested at near, and it may be found that the subject has one of the conditions listed below.

The "A" and "V" Syndromes

Some individuals have a significant difference in muscle balance on up gaze from that on down gaze. If the difference is as much as 20 diopters, it is called either an "A" syndrome or a "V" syndrome. If the eyes are "closer together" (i.e., more eso or less exo) on up gaze, it is an "A," and if they are closer together on down gaze, it is a "V" syndrome. This is one of the reasons why it is so important to check the parallel movements of the two eyes in the various cardinal fields of gaze. For example, one may find an individual who has an exophoria of acceptable degree (i.e., 10 prism diopters or less) in the primary position but on down gaze breaks into a tropia of perhaps 20 or 30 diopters. Since an aviator must maintain conjugate gaze in all directions, it would be unfortunate to let such a person slip by into the flight program.

Noncomitant Squint

The word "squint" in ophthalmology refers to ocular muscle imbalance, not to squinting of the lids. In comitant squint, the angle of deviation is the same in all directions of gaze. In noncomitant squint, the angle is not the same in all directions. In a sense, the "A" and "V" syndromes can be considered noncomitant, but not to the degree of the cases discussed below.

Actions of the Individual Extraocular Muscles.—The bony orbits are shaped like pyramids with their medial walls roughly parallel and their lateral walls, making an angle of 45° with them, or 90° with each other. Five of the six extraocular muscles have their origins in the apex of the orbit and pass forward in various directions to attach to the globe. The sixth muscle, the inferior oblique, arises from inside the rim of the orbit on the nasal side. Each muscle must be considered individually to determine what its actions are (see figure 9–28). The *medial rectus* arises at the apex of the orbit and passes directly forward in the horizontal plane and attaches to the globe. When it contracts, it has only one action, adduction of the globe. Likewise, the *lateral rectus* arises at the apex and passes forward and laterally

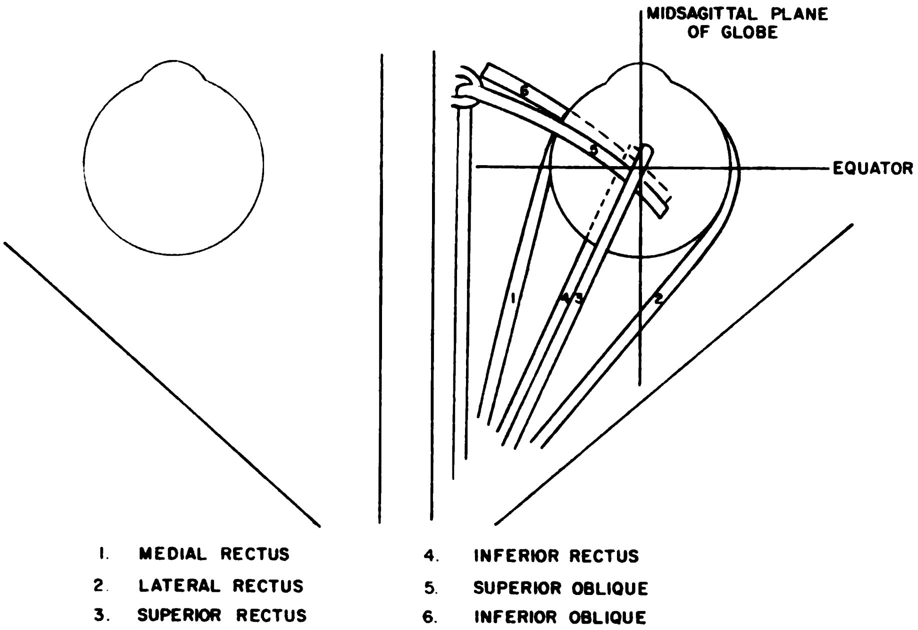


FIGURE 9–28. *Schematic diagram of the locations and attachments to the globe of the extraocular muscles.*

to attach to the globe in the horizontal plane with only one action, abduction. The *superior rectus* arises at the apex and passes forward and laterally to attach to the anterior portion of the globe in the midsagittal plane of the eye. When it contracts, its main action is elevation of the globe, but since it is pulling on an angle, part of its action is to pull medially on the front of the globe, so it is also an adductor. Also, since it pulls medially on the superior portion of the globe, it has a further action of intorsion. The *inferior rectus* runs a course just about parallel to the superior rectus and attaches to the anterior portion of the globe on an angle. It is primarily a depressor of the globe, but since it pulls medially on the front of the globe, it is also an adductor and since it pulls medially on the inferior portion of the globe, it has a further action of extortion. The *inferior oblique* arises just inside the orbital rim on the nasal side and passes laterally and posteriorly below the inferior rectus and attaches to the globe posterior to the equator. When it contracts, it pulls forward and up on the inferior portion of the globe, so its main action is elevation, but it also pulls medially on the posterior part of the globe so that it is an abductor and finally it pulls medially on the inferior of the globe and therefore has a further action of extortion. The *superior oblique* arises at the apex of the orbit and passes forward until its tendon passes through the trochlea, after which it passes laterally and backward beneath the superior rectus to attach to the posterior portion of the globe. Its course after passing through the trochlea roughly parallels that of the inferior oblique. Its pull is chiefly forward on the superior part of the globe so that its main action is depression. It also pulls medially on the posterior part of the globe and therefore it is also an abductor, and since it pulls medially on the superior surface of the globe, it has an action of intortion. These actions are summarized below:

Muscle	*Main action*	*Subsidiary actions*
Medial Rectus	Adduction	
Lateral Rectus	Abduction	
Superior Rectus	Elevation	Adduction, Intortion.
Inferior Rectus	Depression	Adduction, Extortion.
Inferior Oblique	Elevation	Abduction, Extortion.
Superior Oblique	Depression	Abduction, Intortion.

The following generalizations may be helpful in remembering:

(1) Medial and lateral recti have one action each, abduction and adduction.

(2) All "superior muscles" are intorters.

(3) All "inferior muscles" are extorters.

(4) All obliques are abductors.

(5) All vertical recti are adductors.

Field of Action.—The above actions have, of course, been given as they are when the eyes are in the primary position of gaze; that is, looking straight ahead on a horizontal plane. In order to consider what happens when the eye is turned to other positions (see figure 9–28), it is necessary to visualize the right eye turned temporally until the pull of the superior rectus is directly in line with the midsagittal plane of the globe. The superior rectus will then function much more efficiently as an elevator, and it will have no action as an adductor or intorter. Up and right gaze then is said to be the field of action of the right superior rectus, as this is the field of gaze in which it performs its main action best. If the superior rectus is weak, this will also be the field in which the deviation of the eyes will be greatest. Likewise, the inferior rectus functions best as a depressor when the eye looks out and down. The field of action then, for the right inferior rectus is eyes down and right. When the eye is turned in toward the nose, obliques are pulling directly in line with the midsagittal plane of the globe, and this is where they perform their main action best. The right inferior oblique elevates best on upper left gaze, and this is its field of action, and the right superior oblique depresses the eye most efficiently on eyes down and left. These are the fields of actions of these two muscles. In each of these instances, the muscle primarily responsible for the movement must be assisted by a synergist. On eyes upper right, the synergist of the right superior rectus is the right lateral rectus, etc. The fields of gaze of all of the extraocular muscles are summarized on the chart below (figure 9–29). The chart is constructed as though the examiner were facing the patient.

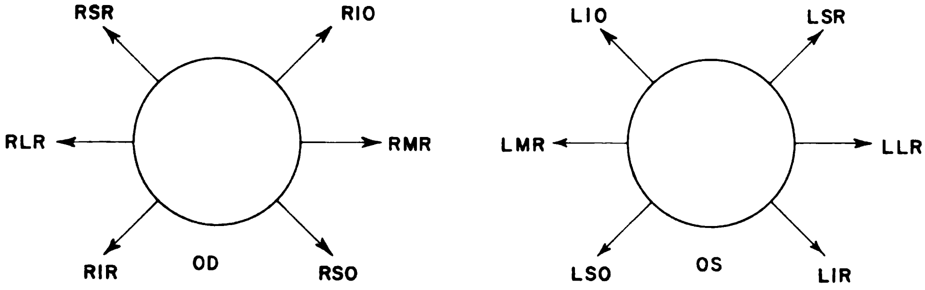


FIGURE 9–29. *Summary of the actions (fields of gaze) of the extraocular muscles, as seen by the examiner.*

Yoke Muscles.—From figure 9–29 above it can be seen that for each of the cardinal fields of gaze there are two muscles involved, one in each eye. When the eyes look directly to the right, this is the field of action of the right lateral rectus and the left medial rectus. On upper right gaze, the two muscles involved are the right superior rectus and the left inferior oblique. On upper left gaze, it is the right inferior oblique and the left superior rectus; on eyes left, it is the right medial rectus and the left lateral rectus; on eyes down and left, it is the right superior oblique and the left inferior rectus; and on eyes down and right, it is the right inferior rectus and left superior oblique. These are shown graphically in figure 9–30.

The whole purpose of including this section on noncomitant squint is to stress the importance to the Flight Surgeon of checking the versions as part of the aviation physical examination. It is entirely possible that an individual could have phorias that measured well within the limits of the standard on the Maddox Rod and yet have an eye that just will not move into one of the cardinal fields of gaze because of a paralysis of one of the ocular muscles. When this happens, the individual must either suppress one eye or have diplopia. In formation flying, particularly, either suppression or diplopia would obviously be hazardous. Not infrequently a young man will apply for flight training knowing that he has a noncomitant squint, but just not realizing that it will be any kind of handicap for him. It may be that his ocular muscle balance is reasonably good in the primary position but when he looks up and left, for example, his right eye will not elevate. Should he be required to fly in formation below and to the right of the leader, he will need to be looking up and left constantly, while maintaining a fairly close interval; and yet, when he looks up and left, he must either have diplopia or suppress one eye, probably the latter. So the Flight Surgeon should always check the versions, or as sometimes said in Navy medical parlance, "take the patient around the horn"; that is, check the position of both eyes in the various cardinal fields of gaze.

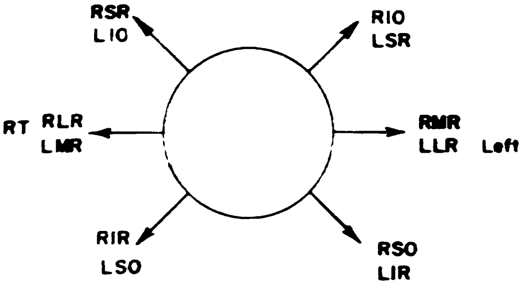


FIGURE 9–30. *Yoke muscles.*

Although it is not necessary to the aviation physical examination, it may be of interest when a case of noncomitant squint is found to determine what muscle or muscles are involved. All one needs do is find the field of gaze in which the deviation is greatest and this will indicate that it is either an overaction of one of the two yoke muscles, or an underaction of the other. As an example, a right hyper that is greatest on upper left gaze may be considered. Upper left gaze is the field of action of the right inferior oblique and the left superior rectus, and since this is a right hyper, it must be either an overaction of the right inferior oblique or an underaction of the left superior rectus.

There are a few simple rules which will help the Flight Surgeon quickly to recall the yoke muscle of any given ocular muscle.

(1) The yoke muscle of a "medial" muscle is a "lateral" muscle and vice versa.

(2) The yoke muscle of a "right eye muscle" is a "left eye muscle" and vice versa.

(3) The yoke muscle of a "superior" muscle is an "inferior" muscle and vice versa.

(4) The yoke muscle of an oblique is a rectus.

(5) The yoke muscle of a vertical rectus muscle is an oblique.

Vicarious Positions of the Head.—These can be classified as head tips, head turns, and head tilts. Actually these positions of the head are frequently combinations of all three but with one of them definitely predominant, depending upon the muscle involved. Whenever a subject appears to be characteristically holding his head in any position other than a relaxed, erect position, the Flight Surgeon should be suspicious of noncomitant squint and insist that such a person sit with his head erect when his phorias are being measured. Such people hold their heads in these positions in order to keep their eyes out of the field of action of the paralyzed muscle, and if they are allowed to maintain that position while phorias are being checked, readings much lower than their true muscle balance will be obtained.

Head Tips. When an individual tips his head backward and looks down his nose, it may mean that he has an "Exo-V" syndrome. It is easier for him to maintain fusion while looking down, so he tips his head back in order to be able to do so. Conversely, if he tips his head forward, it may mean that he has an "Exo-A" syndrome. His muscle balance is better when he looks up, so he tips his head forward in order that the position of his eyes is "up" relative to his head, even when the actual direction of gaze is horizontal.

Head Turns. These are characteristic of palsies of one of the horizontal recti, and the person turns his head so as to get his eyes out of the field of action of the paralyzed muscle. If he turns his head to the right and looks back to his left, he has a palsy of either the right lateral rectus or the left medial rectus. If he turns his head to the left and looks back to his right, he has a palsy of either the right medial rectus or the left lateral rectus.

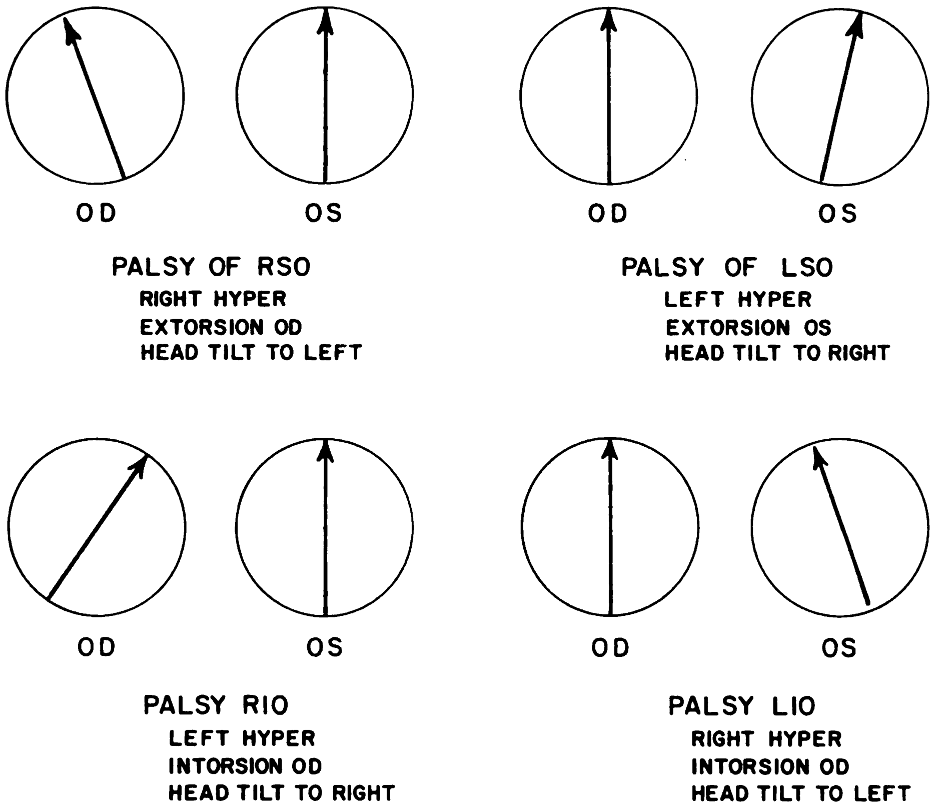
Head Tilts are due to palsies of one of the obliques and can only be explained on the basis of their torsional effect. Every patient who has a weak oblique muscle does not have a head tilt; if a patient has a head tilt due to his eyes it is, however, due to an oblique. In the first example shown in figure 9-31 below, if there is a palsy of the right superior oblique, which is a depressor, there will be a right hyper. It is also an intorter, so if palsied, there will be extorsion of the right eye, and if it is sufficient to cause a head tilt it will be to the left shoulder because the individual will be trying to place the vertical meridian of that right eye in a vertical position. The intorters of the uninvolved left eye will cause it to rotate so that its vertical meridian also remains vertical. (In normal individuals, when the head is tilted to either side, the eyes rotate within limits in order to keep the vertical meridians of both eyes vertical.)

Similarly, if there is a palsy of the left superior oblique, there will be a left hyper and if the torsional effect is great enough, there will be a head tilt to the right. In a palsy of the left inferior oblique there will be a right hyper, and if the palsy is severe enough there will be a head tilt to the left.

Again, it is not necessary for the purpose of the aviation physical examination to determine which muscle or muscles are involved, but if a candidate for a flight program has a vicarious position of the head, noncomitant squint must be considered and, above all, the Flight Surgeon must insist that all examinees hold their heads in a normal erect position while their phorias are checked.

THE ACCOMMODATION-CONVERGENCE REFLEX

Whenever anyone except an uncorrected myope looks from a far point to a near one, three things occur: (1) The eyes converge so that the visual axes of the two eyes both point toward the object of regard, (2) the eyes



Arrows represent 12 O'clock Meridian.

FIGURE 9–31. *Direction of head tilt due to palsy of various extraocular muscles.*

accommodate in order to bring the image of the object into focus, and (3) the pupils constrict to improve the depth of field. The latter item is not of particular interest at this moment. The first two, however, are of great importance in understanding ocular motility. These two phenomena are linked together in all human beings by an inherited reflex so that accommodation stimulates convergence (and vice versa). As a result, most, though not all, hyperopes show esodeviation and most, but not all, myopes show exodeviation. This comes about as follows. A hyperopic eye is usually a small eye; therefore most infants are hyperopic. Hyperopes must accommodate to see at distance, and when they look at near, they must generate three more diopters of accommodation as has been discussed earlier. When a child becomes 2½ to 3 years old, he begins to be interested in the minute details of things around him—his toys, etc.—and he begins to accommodate in order to get a clear image. If he is a high hyperope, this act of accommodation will stimulate his convergence and thereby cause him to develop an esodeviation, quite often an esotropia. If this occurs, it is called accommodative esotropia, and correcting his hyperopia with glasses will either improve or correct his deviation. Of course, some squints are neuromuscular in origin and some have a combined mechanism, and in such cases glasses have less effect.

The myopic child does not have to accommodate; in fact he tries to relax his accommodation as much as possible. Without the stimulus to accommodate, he lacks a stimulus to converge, so that he is likely to develop an exodeviation. If his myopia is corrected with glasses, he will be in the same position as an emmetrope and will have to accommodate for near with his glasses on. This accommodation will stimulate his convergence and improve or correct his exo. These relationships hold throughout life and, as stated above, most hyperopes show at least some eso, and most myopes show at least some exo. In either case correcting the refractive error tends to lessen the deviation. In the case of myopia with exophoria or tropia, it is even possible to overcorrect the myopic error and further stimulate convergence in some cases. Uncommonly, one will find hyperopia associated with exodeviation, and here it is best to give the least amount of hyperopic correction possible in order not to increase the exo by relieving the accommodative effort. Similarly, in a myope with esodeviation, the least amount of minus correction possible should be used in order to avoid stimulating accommodation and thereby stimulating convergence and increasing the eso.

It is possible to treat purely accommodative esotropia medically and thus to avoid surgery and even glasses. To do this, strong miotics (DFP or Phospholine Iodide) are used. The ciliary muscle undergoes spastic paralysis, and since it is already accommodating maximally, there is no further stimulus to accommodate, and therefore no stimulus to converge. The pinpoint pupil helps to give a clear image. The results are sometimes dramatic. A drop of DFP or Phospholine may cause the eyes to become straight within a few hours and the effect can be maintained by a drop every other day. The "accommodation" produced by the miotic does not produce convergence because its action is peripheral (at the site of the muscle) and there is no effort of accommodation. Cycloplegics, on the other hand, are unpredictable in their effect on squint. Sometimes they improve a squint; at other times, they appear to make it worse.

DEPTH PERCEPTION

There has never been a really good test for depth perception. The Verhoeff Depth Perception test, while one of the best, only tests one clue to depth perception and there are many clues to this facility. Some of them are not even visual, although the nonvisual clues come into play more when there is dim light and the visual clues are restricted. Echoes, for example, influence perception of silhouettes seen on a very dark night. The interest here, however, is only in the visual clues. Each individual uses all of the clues mentioned below throughout his waking life, and does so without being conscious of it. Some of them are very, very simple clues, yet they play an important part in the sense of spatial relationships. The clues to depth perception will be divided into monocular clues, clues that can be either monocular or binocular, and binocular clues.

Monocular Clues

1. *Interposition of Objects.* The person who sees a house and a tree, with part of the tree obscured by the house, knows the tree is behind the house. This seems hardly worthy of mention, yet everyone uses this clue constantly in his total perception of his surroundings.

2. *Convergence of Parallel Lines in the Distance* (*Perspective*). Everyone is familiar with the way railroad tracks appear to approach each other toward the horizon even though they are known to remain parallel. Even without the familiar railroad tracks, there are lines in every landscape which approach each other in the distance and add to the perception of spatial relationships. Artists, of course, are well-versed in the monocular clues to depth perception, and they use all of them, particularly this one, to create a sense of depth in a picture painted on a flat surface.

3. *Motion Parallax.* Motion parallax refers to the apparent relative motion of stationary objects as the observer moves across the landscape. Near objects appear to move backward to the path of motion and far ones seem to move with it.

4. *Relative Size of Familiar Objects.* If one perceives two aircraft of the same type and the image of one is smaller, he immediately assumes that it is farther away. If the images of two objects are about the same size and the viewer is familiar with these two objects so that he knows they are *actually* about the same size, he knows that they are both about the same distance

away from him. If he is unfamiliar with the actual size of the objects, however, this clue does no good and can even be misleading.

5. *Increasing Size of Objects.* If the image of an object is increasing in size, the observer knows that it is approaching and can also judge its speed by how fast its image is expanding. The same is true for decreasing size of images of objects going away.

6. *Color and Haze in the Distance.* Color and haze in the distance are affected by the amount of water vapor and impurities in the air and are much more noticeable in the eastern half of the United States than in the dry, pure air in the West. Objects in the distance take on a bluish color and are hazy in appearance. This can be misleading to an easterner traveling in the West for the first time. Mountains look closer than they really are because of the clarity of the atmosphere.

7. *Lights and Shadows.* These are very subtle clues but very important to the sense of depth. Relative position of objects can be determined by the manner in which their shadows fall. Light from above falls on the top of a protuberance and on the bottom of a depression. If some of the recent pictures of the moon are viewed, right side up, the craters look like craters. If the pictures are inverted, the craters look like hillocks.

Clues That Can Be Monocular or Binocular

1. *Accommodation.* When one focuses on an object, his brain receives proprioceptive sensations from the ciliary muscle as to just how much he is having to accommodate, and his overall sense of depth is thereby increased. This clue is useful only within 20 feet. Normally, it is a binocular clue, but should effective vision in one eye be lost, the clue will still function with the remaining eye.

2. *Convergence.* Proprioceptive sensations are also received from the extraocular muscles which give a reference as to how far away the object of regard is. Again, this is normally a binocular clue, but if vision in one eye has been lost, both eyes still converge on a near object.

Binocular Clues

1. *Physiological Diplopia.* If the average individual is asked if he has ever had diplopia, the chances are he will say no. Yet everyone has diplopia most of the time. The following experiment demonstrates this.

Extend one arm out as far as you can and hold up your thumb. Then extend the other arm with thumb up about half way to the first. Look at the near thumb and if you have normal fusion, you will see two images of the far thumb. This is physiological diplopia. Look at the far thumb and you will see two images of the near thumb. Look at the near thumb and close your right eye. The right-hand image of the far thumb will disappear; this is uncrossed diplopia. Look at the far thumb and close your right eye. The left-hand image of the near thumb disappears; this is crossed diplopia. Physiological diplopia is one of the clues to depth perception. Occasionally it turns up as an ocular complaint, when an individual notices his physiological diplopia by accident. He may come to the Flight Surgeon feeling quite concerned and relate that he was driving down the road and suddenly he noticed that he was seeing two images of some object hanging from his rear view mirror. The simple demonstration with the thumbs described above will reassure him.

2. *Stereopsis.* Stereopsis is the ability to fuse the slightly dissimilar images seen by the two eyes and to achieve a sense of depth. The images must be slightly dissimilar or no stereopsis occurs. In other words, slightly disparate retinal elements must be stimulated to achieve depth and if they are too dissimilar, diplopia results. As a demonstration, do the exercise with the thumbs again, only this time slowly bring the far thumb forward. Just before it reaches the near thumb, there is no further diplopia and the two thumbs are seen in stereoscopic view. The same is true when one looks at the far thumb and moves the near one slowly out to it. This brings up the subject of the *horopter* and *Pannum's Fusional Area.* The horopter is a curved plane in space each point of which stimulates exactly corresponding retinal elements. Therefore, there is no stereopsis of objects situated on the horopter. The distance of the horopter varies with the point of fixation through which it must pass, but its shape remains the same (see figure 9–32). Objects situated on the horopter appear to be situated on a frontal plane. On either side of the horopter there is an area where any object would stimulate slightly dissimilar retinal elements. It is in this area, called Pannum's Fusional Area, that stereopsis occurs. Closer in, and farther out, there is physiological diplopia.

The Verhoeff Depth Perception Test

The Verhoeff Depth Perception testing device consists of a small black box 2 x 4 x 8 inches with a small lighted aperture through which three small

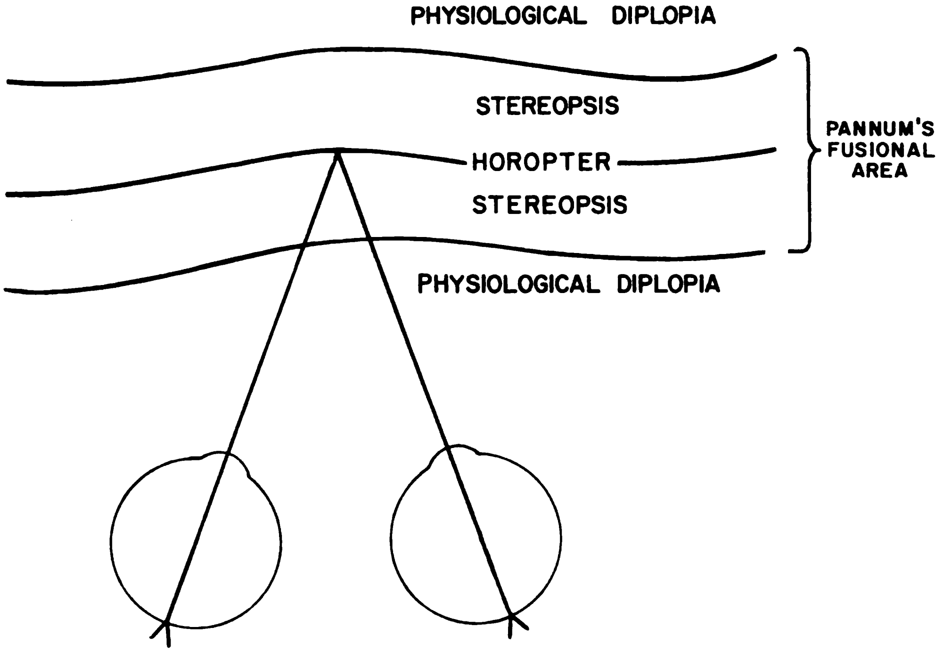


FIGURE 9–32. *Stereoscopic vision.*

posts can be displayed. There are two possible positions for each post, either forward or back. Four different combinations of post settings are possible and by inverting the box, this is increased to eight. In each setting one post is out of line with the other two and the examinee is asked to name the post that is out of line and tell whether it is forward or back. (i.e., right forward," "middle back," etc.). If he calls eight out of eight correctly, he is passed. Should the examinee miss one or more on the first run, he is given two more runs and must score 16 correct out of 16 to pass.

Consideration of this device for a moment and enumeration of the various clues to depth perception listed above, will make it readily apparent that it tests only one clue, stereopsis. In fact, the posts are constructed of varying width in order to rule out size of the images as a clue. The test must be given either in a dark room or in a diffusely lighted room in order to prevent a shadow of the top of the aperture from falling on the posts; otherwise, the test can be passed with ease, even by a person with monocular vision. Also the test must be given at a measured distance of one meter. If this is moved in to 80 centimeters from the subject, it again becomes so easy that almost everyone can pass because the angle of stereopsis becomes greater.

As stated above, the Verhoeff does not test depth perception, it only tests stereopsis. There has never been a really good test for depth perception. In the final analysis, the real test for this faculty is a student pilot's ability to fly successfully, particularly to land his aircraft on a carrier deck, or even on a concrete runway, and to fly a good tight formation maintaining the proper position and attitude while following the flight leader through various maneuvers. Although stereopsis is not the whole of depth perception, as has been shown, it is evident that a military pilot, at least, cannot continue to perform the intricate maneuvers required of him if he is deficient in his ability with any of the clues to depth. So even though the Verhoeff does not test depth perception, it is considered to be a good test. It is a good test because it is highly critical, and it is important that military pilots have adequate stereopsis. If a candidate passes the Verhoeff, it is felt that he does have adequate stereopsis. If he does not pass the Verhoeff, the Bureau of Medicine and Surgery will consider recommendations for a waiver providing the candidate can pass some other stereoscopic tests such as the Armed Forces Vision Tester.

The Armed Forces Vision Tester

The Armed Forces Vision Tester can be used to test visual acuity, muscle balance, and stereopsis. It has many advantages and is especially useful in testing stereopsis. For this, either chart 5 or 5A can be used. Each of these charts has six groups (designated A through F) of three rows of circles, five circles to a row. In each row, one circle should stand out from the chart closer to the examinee and he is asked to name that circle. The test becomes more difficult as one progresses from A to F. In order to pass, the examinee should make no errors in groups A through D. The test seems to have about the desired degree of difficulty.

At the Naval Aerospace Medical Institute, an effort has been made to study in detail those candidates who pass all of the routine eye exams and yet fail the Verhoeff. This has been done from both the ophthalmological and psychiatric standpoints. Preliminary investigations indicate that failure to perform satisfactorily on the Verhoeff may be attributed to: (1) Small-angle tropias previously misdiagnosed as phorias. These were in the range of one to three diopters. (2) Excessive, and even borderline refractive error. Those Service Group III pilots who are required to wear glasses will probably have to wear them in order to pass the test. (3) Variable phorias. The individual is found to have a phoria within the standards in the examining room but because of failure on the Verhoeff, he is retested several times and found to have a variable phoria that for the most part exceeds the minimums. (4) Anisemmetropia, either spherical or cylindrical, of a degree that would not be considered significant elsewhere. (5) Ambivalence about flying. Some individuals definitely feel they want to fly in the sense that they would like to see themselves in the role of a pilot, yet they have serious doubts concerning their ability to achieve this goal. A psychophysiologic reaction occurs which blocks their ability to pass the test. (6) Subclinical psychopathology. It is said that many schizophrenics see the world as flat. Minor emotional problems and even more serious ones that have been well compensated for may be just severe enough to cause a failure on the Verhoeff. One candidate who had failed the Verhoeff stated that when he looked in a mirror he had always thought of his image as being on the surface of the mirror rather than behind it. (7) Malingering. These are easy to detect by the excessive show of effort when taking the test and by the false and inappropriate demonstration of emotion upon being told that they have again failed the test. However, it is one thing to be satisfied in one's own mind that a candidate is malingering and quite another to prove it. In fact, it is not necessary to prove it. Malingerers are not desired in aviation. It is true that it would be better for the individual's character if he would drop from training at his own request, or turn in his wings, in the case of a designated aviator, but if he cannot bring himself to do so, there is no sense belaboring him. The Flight Surgeon should simply find him not physically qualified by reason of failure to pass the depth perception test (or whatever the failure may be). The word "malingering" should never be entered into a record for any reason unless the proof is incontrovertible, and such instances are rare indeed.

It is unwise also, to ascribe failure on any part of the physical examination to psychogenic causes unless it can be substantiated by positive findings on psychiatric examination, and then only if it is necessary in order to make proper disposition of the case. Diagnoses of psychogenic disorders should not be made on the basis of exclusion of organic findings. Such a thing is unfair to the patient or subject since "exclusion of organic findings" may actually represent failure to find pathology that is present.

VISUAL FIELDS

Testing of the visual fields is not a required part of the routine aviation physical examination except by confrontation. Admittedly, confrontation is an exceedingly gross method and is unlikely to disclose abnormalities unless they are relatively large. However, visual field testing, if properly and meticulously done, is so time-consuming that it is simply not practicable to perform it routinely. Certainly visual field testing should be done on candidates for astronaut training and on others selected for missions of critical importance. If there is anything in the history suggestive of constriction or scotomata, then careful and complete fields must be done on any examinee. This holds also for any case in which there are suspicious fundal findings.

The perimeter is the better instrument to test for peripheral constriction of the field, whereas the tangent screen is much the better for plotting scotomata. In fact, "peripheral fields" refer to the visual fields as plotted on the perimeter, and "central fields" refer to the fields as determined on the tangent screen. Both are needed to evaluate a case properly. Every physician has been exposed, at least a few times, to the use of these two testing devices, so there is no need for a description of the apparatus here. Below are a few words on their proper use. (1) The examiner should remember to blindfold one eye and try to arrange the elastic holding the eyepatch in place so that it does not press on the lids or brow of the eye to be tested. (2) If the subject wears glasses, they should be used while testing central fields. It is better to remove them while testing peripheral fields as the rims get in the way. (3) The purpose of the test should be fully explained to the subject to make sure he knows what is expected. (4) The examiner should try to stand so as to watch the subject's eyes and make certain he keeps looking at the fixation point. (5) If subject has heavy and protuberant brows, the examiner should elevate them with his fingers while testing the superior field. (6) The smallest target that the examinee can see should be used. (7) To chart the periphery of the field and the edge of any scotoma the target is moved from the unseen to the seen. (8) If a target on a wand is used, the target should be kept ahead of the wand. That is, make sure the target enters the "seen" portion before the wand. (9) Care should be taken to record the name, date, the size of the target (either in millimeters or degrees), and the distance at which the test was performed. (10) If hysteria is suspected, the tangent screen examination should be performed at different distances, but the diameter of the target should be doubled if the distance is doubled. Getting the same size field under these circumstances, at say one meter and two meters, is referred to as gun-barrel vision and is diagnostic of hysteria. The examiner should bear in mind that individuals with organic lesions can also demonstrate a hysterical field. (11) If a field is found to be cut, it is well to test with different size targets.

The Visual Pathway and Interpretation of Visual Fields

Most physicians are familiar with the visual pathway but perhaps a brief review is in order. The pathway begins, of course, with the rods and cones of the retina, and after two synapses in the retina itself, goes via the nerve fiber layer toward the optic discs. Since there are only cones in the macula, and each single cone is represented by its own nerve fiber, there is a thick band of nerve fibers running from the macula to the optic disc, called the papulomacular bundle. It then becomes necessary for the nerve fibers in the surrounding retina to arch around this bundle as shown in figure 9–33. There is a horizontal raphe separating fibers from the superior and inferior retina. Lesions anywhere in the retina produce scotoma but if they are in the periphery, they produce small scotomata that are likely to be of little consequence. Lesions near the disc, however, are likely to involve the nerve fiber layer and if so, they cause "sector defects." If a lesion involves temporal fibers near the disc, it is likely to produce a "sector defect" that arches over or under the point of fixation, in which case it is called an "arcuate scotoma" (see figure 9–34). Glaucoma can cause such a scotoma. Chorioretinitis near the disc can also produce a sector defect which may be arcuate, if the lesion is temporal. This is called "Jensen's Chorioretinitis Juxtapapillaris." In the optic nerve, the fibers from various parts of the retina maintain roughly their same relative position. In the optic chiasm, the nasal fibers cross; the temporal ones do not. It should be noted in passing that the chiasm is not just a simple crossing. Some temporal fibers enter the chiasm and

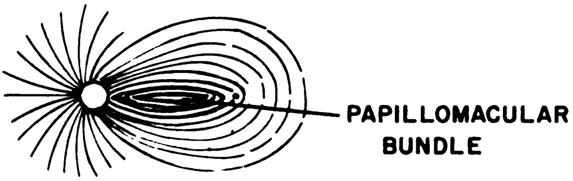
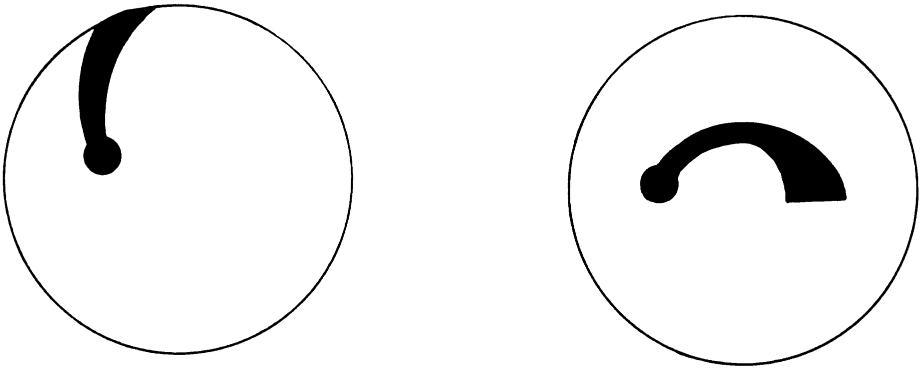


FIGURE 9–33. *Distribution of nerve fibers.*



SECTOR DEFECT

ARCUATE SCOTOMA

FIGURE 9–34. *Sector defects.*

then come back out. Some nasal fibers pass into the tract on that side and then bend back and cross. A detailed description is not needed here except to say that some chiasmal lesions can produce apparently bizarre field defects because of this. From the chiasm, the ipsilateral temporal fibers and the contralateral nasal fibers proceed via the optic tracts to the lateral geniculate body where there is a synapse. From the lateral geniculate body the pathway goes via the optic radiations to the calcarine cortex (figure 9–35).

Sections of the optic nerve produce, of course, a blind eye. Partial interruption of the nerve produces monocular field defects. Sections of the chiasm interrupt the nasal fibers from both eyes and thereby produce bitemporal hemianopsia. Since most lesions involving the chiasm come up from below, the defect usually begins as a bitemporal superior quadrantanopsia.

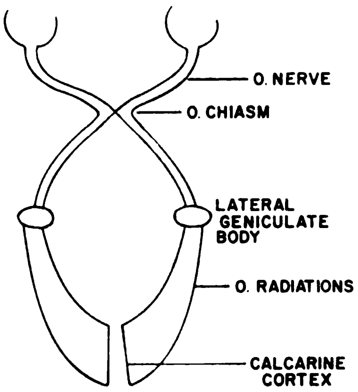


FIGURE 9–35. *Visual pathways.*

Lesions anywhere in the tracts, geniculate bodies, radiations, and calcarine fissures involve nasal fibers from one side and temporal fibers from the other and produce homonymous hemianopsia. In the optic tracts, the fibers from corresponding retinal elements in the two eyes are not in juxtaposition to each other, but as they proceed back toward the calcarine cortex, they tend to run more and more closely together. This produces the phenomenon of "congruity" of field defects; that is, the nearer the lesion is to the cortex, the more congruous will the field defects be.

If one finds a field defect, then the fields should be tested with targets of different size. If the field or defect is very nearly the same (within 2° or 3°) size when charted with a 10 millimeter test object as with a 2 millimeter test object, the defect is said to have a "steep" edge. If the field is significantly larger with the 10 millimeter object, the defect is said to have a "sloping" edge. A steep edge is indicative, though not diagnostic, of a vascular lesion, and a sloping edge is indicative of an expanding lesion. It can readily be seen why this is so. In a vascular lesion, the blood supply to the brain tissue is adequate up to a fairly well-demarcated line, with healthy tissue on one side and nonfunctioning tissue on the other. In an expanding lesion, the pressure is greatest immediately adjacent to the mass and lessens farther away so that there tends to be a gradual and progressive destruction.

THE PHARMACODYNAMICS OF THE PUPIL

Innervation

The sphincter muscle of the pupil is innervated by the parasympathetic system, the dilator by the sympathetic system. The stimulus for the parasympathetic system is light, and the afferent pathway is the same as the visual fibers until a point just proximal to the lateral geniculate body. The pupillary fibers cannot be distinguished from the visual fibers by any staining method. The pupillary fibers decussate in the same way as do the visual fibers. Just before the visual fibers enter the lateral geniculate body, the pupillary fibers leave and proceed to the pretectal area where there is another decussation and the pretectal nucleus. After a synapse in the pretectal nucleus, the fibers proceed to the Edinger-Westphal nucleus of the third nerve. The efferent pathway goes via the third nerve and then its inferior portion, thence to the ciliary ganglion where there is a synapse. The postganglionic fibers go via the short posterior ciliary nerves and finally reach the sphincter of the pupil (see figure 9-36). Section of the optic nerve then produces a blind eye but the pupil will still respond to consensual stimulation of the fellow eye because of the chiasmal decussation and also the pretectal decussation. The eye will not, however, respond to direct stimulation. Section of the chiasm produces, not only a bitemporal hemianopsia, but also a bitemporal hemianopic pupillary response. That is, if one shines a light in so that it strikes the nasal retina of either eye, there will be very little response in either eye (possibly some slight reaction due to scatter). However, if one shines a light so that it strikes the temporal retina of either eye, normal direct and consensual response will be obtained.

If there is a section of the optic tract prior to the point at which the pupillary fibers leave the visual fibers, there is a homonymous hemianopsia and a homonymous hemianopic pupillary response. If one stimulates the temporal retina on that side or the nasal retina on the opposite side, there is little or no pupillary response, but if one stimulates the nasal retina on that side or the temporal retina on the opposite side, a normal pupillary response will result.

Lesions of the visual fibers after the pupillary fibers have separated do not change the pupillary reaction. Lesions of the pupillary fibers after they have separated from the visual, do not affect vision, but do produce homonymous hemianopic pupillary responses.

The stimulus for the sympathetic system is not the absence of light but rather it is emotion, such as fear, pain, excitement, even love. The afferent pathway is via the sensory nerve involved in the case of pain or via a special

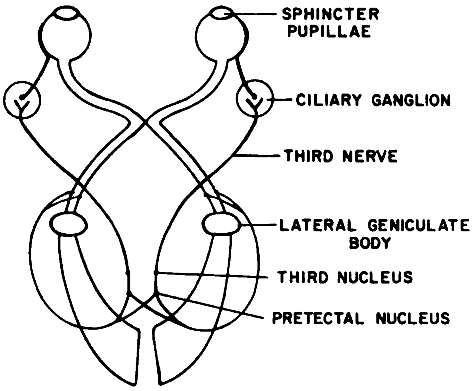


FIGURE 9-36. *The parasympathetic system.*

pathway from the frontal lobe in the case of emotion. The nucleus is thought to be somewhere in the pons. From the nucleus the preganglionic fibers proceed down the cord to the lower cervical region, exit the cord and synapse in the superior cervical ganglion. Then the fibers run in the carotid plexus and to the orbit. In the orbit some fibers go to the orbital muscle; some enter the ciliary ganglion (but do not synapse) and reach the pupil via the short posterior ciliary nerves; others proceed up to supply Mueller's muscle in the upper lid, and still others proceed further and supply the sweat glands of the skin of the brow. A distal lesion then can produce simple anhydrosis of the brow. If the lesion is a little lower, it can produce anhydrosis, and ptosis due to the loss of tone of Mueller's muscle. Still further down a lesion can produce anidrosis, ptosis, and miosis, and if the lesion also involves the fibers to the orbital muscle the result is anidrosis, ptosis, miosis, and enophthalmos (a full-blown Harness syndrome).

Pharmacology

In the neurohumoral mechanism for the transmission of nerve impulses there is in every synapse an effector substance and an antagonist which destroys the effector substance in order to prepare for the next impulse. In the parasympathetic system, the effector substance is acetylcholine both in the synapse between the pre- and postganglionic fibers and between the postganglionic fiber and the effector organ. Its antagonist is cholinesterase. Acetylcholine is the effector substance in the voluntary muscular system, and also in the sympathetic system in the synapse between the pre- and postganglionic fibers. In the synapse between the postganglionic fiber and the effector organ in the sympathetic system the effector substance is sympathin. Its antagonist is amine oxidase.

Parasympathetic depressors cause dilation of the pupil by producing flaccid paralysis of the sphincter muscle. The pupil becomes dilated from the normal tone of the dilator muscle. The parasympathetic depressors used in ophthalmology all exert their depressant effect on the motor end plate of the muscle fibers making them insensitive—or less sensitive—to the action of acetylcholine. The ciliary muscle is also innervated by the parasympathetic system so that these drugs also produce flaccid paralysis of this muscle in the same way. For this reason such drugs are called cycloplegics. They are used for dilation when one wants to put the eye at rest as for refraction or in the treatment of iritis. Among the parasympathetic depressors are atropine, homatropine, scopolamine, Cyclogyl Hydrochloride and Mydriacyl. Dosages are given below.

Excitatory parasympathetic drugs cause constriction of the pupil and they are divided into two groups. One group consists of the cholinesterase inhibitors. By blocking the action of cholinesterase, they allow the acetylcholine which is present to act continuously on the motor end plates of the sphincter. Some of these drugs are: Eserine, physostigmine, prostigmine, DFP (diisopropylfluorophosphate) and Phospholine Iodide. The latter two drugs were discovered by researchers working on the nerve gases and are closely related to these compounds. They are powerful miotics. The second group of parasympathomimetic drugs has its action directly on the motor end plates of the sphincter. They are pilocarpine, Doryl, Mecholyl, muscarine, and acetylcholine.

Sympathetic depressors are not generally used for topical administration in ophthalmology. They act by depressing the sensitivity of the motor end plate of the dilator muscle to the action of sympathin. Two of them are Dibenamine and Priscoline.

Sympathetic excitatory drugs (sympathomimetics) are also divided into two groups. Cocaine is an amine oxidase inhibiting agent. It produces dilation of the pupil by allowing the sympathin which is present to act continuously on the motor end plate of the dilator muscle. It occupies the same place in the sympathetic system that the cholinesterase inhibitors occupy in the parasympathetic. Although cocaine has been used in the past for mydriasis and is still used by some for topical anesthesia, it is not recommended. It is a highly toxic drug and in a rare patient one drop may cause cardiac arrest. It is of interest that topical cocaine not only causes mydriasis, but also causes elevation of the lid by stimulation of Mueller's muscle. It has been used by actors to simulate fright because it produces big pupils and wide-open, staring eyes, and of course, it is a sympathetic discharge that produces this appearance in actual fright. The other group of sympathomimetic drugs has its action directly on the motor end plates of the dilator. They are adrenalin, Neo-Synephrine, Paredrine and ephedrine, and are used when one desires mydriasis and not cycloplegia. Ten percent Neo-Synephrine is particularly useful in this regard, when one merely wants to open up the pupil for examination of the fundus or lens. The mydriatics are more easily overcome by miotics than the cycloplegics in general so the pupil can be constricted when the examination has been completed.

	DOSAGES
Dilators:	
Cycloplegics:	
Atropine * Solution 1 percent	1 to 3 times a day for iritis.
Atropine Ointment ½ or 1 percent.	3 times a day for 3 days for refraction, infants. Used at each dressing change postoperatively.
Homatropine 4 or 5 percent Solution.	1 or 2 drops 2 or 3 times for refraction.
Scopolamine ¼ percent	1 to 3 times a day.
Cyclogyl ½ percent	1 or 2 drops 2 or 3 times 10 minutes apart for refraction, small children.
Cyclogyl 1 percent	1 or 2 drops 2 or 3 times 10 minutes apart for refraction.
Mydriacyl ½ percent	1 or 2 drops 2 or 3 times 10 minutes apart for refraction, small children.
Mydriacyl 1 percent	1 or 2 drops 2 or 3 times 10 minutes apart for refraction.
Mydriatics:	
Neo-Synephrine 10 percent	1 or 2 drops 2 to 5 times as necessary.
Ephedrine bitartrate, HCl, Borate, 1 and 2 percent.	In conjunction with miotics in glaucoma.
Adrenaline 1:1,000 with procaine or Xylocaine, and Atropine 1 percent equal parts.	2 to 5 minims injected subtenons to break up synechiae.
Constrictors—Miotics:	
Cholinesterase Inhibitors:	
Eserine ointment 1 percent	Used at H.S. in glaucoma.
DFP 0.06 and 0.125 percent	Once a day or once every other day in glaucoma and in the medical treatment of esotropia.
Phospholine Iodide 0.06, 0.125 and 0.25 percent.	Once every other day to b.i.d. in glaucoma and in medical treatment of esotropia.
Action Directly on Motor End Plate of Sphincter:	
Pilocarpine 1 to 10 percent	3 to 5 times a day in glaucoma. 1 and 2 percent used 1 to 3 times for constricting pupil following dilation.
Mecholyl 20 percent	In glaucoma t.i.d., usually in conjunction with other drugs.

*Never use atropine drops in children. It is possible for them to absorb a toxic and even a fatal dose. If atropine is desired, use ointment, preferably ½ percent t.i.d. for 3 days prior to the refraction. Nothing on the day of the examination. Instruct parents to discontinue the medication if flushing occurs.

Cyclogyl can cause hallucinations in children but the effect is short lasting and is not a contraindication to its use unless the child is known to have reacted previously. Cyclogyl can also cause light-headedness and in rare instances syncope in adults. Again, this is not a contraindication unless the patient is known to have had symptoms from the drug previously.

Atropine causes cycloplegia that lasts two weeks in the normal healthy eye, scopolamine, about one week. When medical personnel present with sudden onset of dilated pupils and more especially with one pupil dilated, the most likely and obvious thing is that they have contaminated their fingers and then their eyes while administering one of these drugs.

The effects of Homatropine lasts 2 to 3 days; cyclogyl 36 hours; mydriacil about 4 hours. Aviators should not be allowed to fly until all effects of cycloplegia wear off and not for a minimum of 3 days.

OCULAR TRAUMA

This topic will be organized from an anatomical standpoint for the most part, beginning with the lids and progressing back into the orbit, bearing in mind that various types of trauma such as blunt trauma, sharp trauma, chemical and radiation trauma may affect most parts of the eye and adnexa. Initial treatment or first aid will be given for the benefit of the Flight Surgeon who plans to evacuate the patient to a hospital and is in a position to do so. The definitive treatment will then be discussed for the Flight Surgeon who desires to treat the case himself or is in an isolated area where he must provide the treatment.

The Lids

Contusions of the lids constitute the most common eye injuries. The chief consideration here is to check for other injuries. A recently acquired ecchymotic eye is acutely tender and frequently the swelling is so great as to obstruct observation. One cannot be squeamish about producing a little pain, although, naturally one should be as gentle as possible. It is necessary however, to pry the lids open, examine the anterior chamber, the pupil, the fundus, and the motions of the eyeball as completely as possible. Care must be exercised not to increase any damage that has been done. One should palpate the rim of the orbit for any sign of fracture. It is also possible to have a fracture of the floor of the orbit without a fracture of the rim. This is called a blowout fracture, and is an injury that is often missed for several days, when there is a lot of ecchymosis. Where the floor has been more or less demolished, in some cases the eyeball may drop as orbital contents fall into the maxillary sinus, in which case the injury is easy to recognize. In other cases the globe does not drop since it is held in place by the suspensory ligament of Lockwood. The defect can be seen on X-ray, however. In other instances, there may be a crack in the floor and some of the orbital fascia may be incarcerated in the fracture line. These fractures may not be seen on X-ray but if the orbital fascia is incarcerated as described, the eyeball will be limited in its motion. The injured eye will act as though it were being held by a thumb forceps and it will neither elevate nor depress with the fellow eye. First aid in these cases is to cover the patient with a systemic antibiotic to prevent an orbititis from developing from the bacteria in the sinus and evacuate the patient to a hospital. Briefly, there are two ways of handling these cases. The maxillary sinus may be approached through a Caldwell-Luc and the roof of the sinus (floor of the orbit) packed back into position, or an incision may be made at the base of the lower lid (outside the rim so as to be outside the septum orbitale of the lid) carrying it down through the periosteum. The periosteum is then elevated with a periosteal elevator to expose the fracture, the orbital contents are teased out of the fracture line, and the defect bridged over with tantalum mesh or other inert material.

Lacerations of the Lids.—Lacerations about the eye usually occur over the bony prominences of the brow and cheek. These are, of course, of no consequence and can be sutured in whatever manner desired. Even lacerations of the lids themselves constitute no special problem unless they involve the lid margins, especially the lower lid margin and even more especially the lid margin near the inner canthus. The lid glands produce an oily secretion which covers the lid margins and keeps the tears back within the conjunctival sac. If one sutures a laceration of the lid margin with simple interrupted sutures, a tiny "V-defect" will almost always ensue (see figure 9–37). The oily film will now be interrupted with the result that the tears continuously spill down the cheek. Tears spilling from the eye constitute an epiphora. Lacerations of the lid margins should, if possible, be sutured in the hospital and the first aid would include: (1) Ophthalmic antibiotic ointment, (2) sterile dressing, (3) tetanus toxoid. It is important to give a tetanus booster

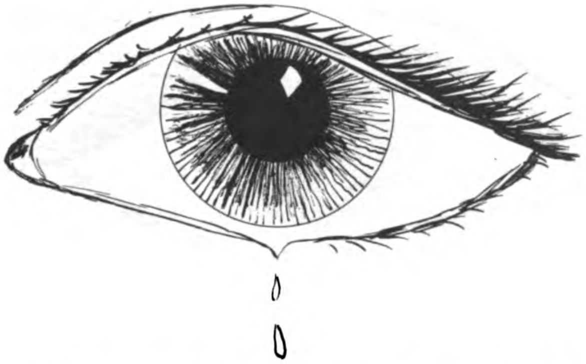
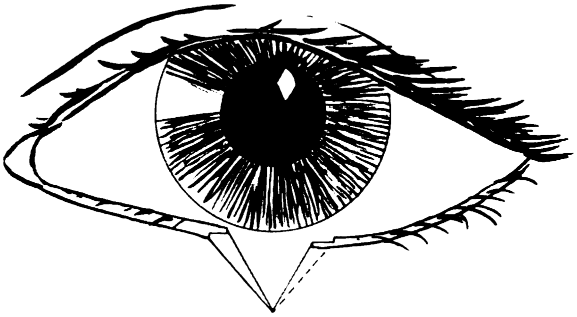


FIGURE 9–37. *V-defect with epiphora following improper closure of laceration of lid margin.*

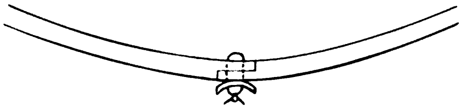
in any lacerating or penetrating injury about the eye because of the proximity of the brain.

There are two ways of suturing a laceration of the lid margin. The most effective way is by a "lid-halving procedure." If you look at the margin of a lid you will note a line which divides the margin into two halves. This is called the "grey line." To do a lid-halving, one first makes an incision through the grey line for about 2 mm on each side of the laceration. This divides the lid into skin and orbicularis muscle in the outer leaf and tarsus and conjunctiva in the inner leaf. Now one excises 2 mm of the outer leaf on one side of the laceration and 2 mm of the inner leaf on the other so that the two sides can be brought together and overlapped. A fairly strong (4–0 double-armed silk) suture is passed through both leaves from the posterior surface, then through a small piece of soft rubber catheter (to prevent the suture from cutting back through the lid) and tied (see figure 9–38). The skin is closed further with interrupted sutures of smaller silk as desired. The conjunctival surface need not be sutured. The skin sutures can be removed in 3 to 5 days, but the through and through 4–0 suture should be left in for about 10 days to 2 weeks.

Lid-halving might be a little more surgery than the Flight Surgeon would want to attempt unless he had had previous experience with it. Another method that produces good result in 90 percent or better of cases is the figure-of-8 suture. To do this, a single-armed 4–0 (approximately) silk suture is used and one begins by taking a superficial bite on one side of the laceration, passing the needle in through the grey line, and bringing it out through the cut edge. Next, a bite is taken more toward the apex of the laceration on



SKIN AND ORBICULARIS HAVE BEEN EXCISED ON ONE SIDE, TARSUS AND CONJUNCTIVA ON THE OTHER



OVERLAP AND CLOSURE. TIE OVER PIECE OF SOFT RUBBER CATHETER

FIGURE 9–38. *Lid-halving procedure.*

the opposite side, passing the needle in through the cut edge and also out through the cut edge. A similar bite is then taken on the original side, and the needle passed in through the cut edge and brought out in the grey line on the side of the laceration opposite the very first bite and the suture tied (see figure 9–39). The skin is closed with interrupted sutures as needed. This is a fairly simple procedure and is successful in many cases. If a V-defect results, it can be corrected as an elective procedure using lid-halving at a later date.

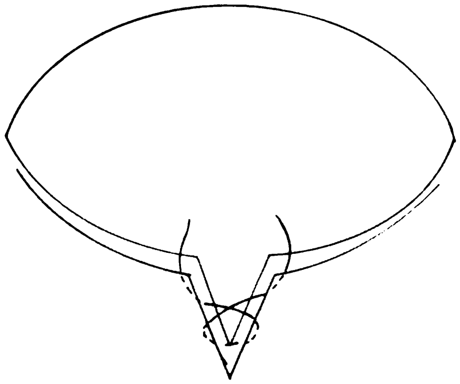


FIGURE 9–39. *Figure-of-8 suture.*

If there is a laceration of the margin between the punctum lacrimale of the lower lid and the inner canthus, then the canaliculus will have been cut and it must be reestablished, else a constant epiphora will result. This is a difficult procedure when performed under optimum conditions by experienced personnel, and such cases should be evacuated to the hospital even if it will take 2 or 3 days for the patient to get there. The first aid should be as above: antibiotic, sterile dressing, and tetanus booster. If the upper canaliculus is cut, it is not of too great importance since the lower canaliculus handles nearly all of the drainage of tears. There are several ways to handle lacerations involving the lower canaliculus (and the upper, if it turns out to be easy). One way is to pass a piece of 26-gauge polyethylene tubing in through the punctum and out through the cut end. Find the other cut end (this is the difficult part) and pass the tube into it and on down toward the nose. Suture it in place and close the rest of the wound. Leave in place two to three weeks. Another way is the "pigtail" probe method. Bend the end of a probe into a half circle in a plane perpendicular to the rest of the probe. Pass this probe into the upper punctum, around through the nasolacrimal sac, and back out through the lower canaliculus to its cut end, then into the other cut end and out through the lower punctum. Tie a suture to this and drag it back through. Disengage the probe and tie the two ends of the suture together so that it serves as a sort of splint.

The Conjunctiva

Contusions.—Subconjunctival hemorrhages are of no importance if there is no other injury. They take two weeks to absorb. Not infrequently, one sees subconjunctival hemorrhage without a history of trauma. The most likely cause is that the patient has been coughing, sneezing, or vomiting and thereby ruptured a small conjunctival vessel. The incident may have happened a week before and if the rupture was in the superior fornix, it would not have been noted until it filtered down by gravity and appeared in the interpalpebral area.

Lacerations of the Conjunctiva.—These are also of no great concern provided there is no other injury. If the pearly white sclera can be seen and there is no retained foreign body, the conjunctiva can be closed with a running suture of either silk or gut using procaine or Xylocaine subconjunctivally. Sometimes it is possible to suture the conjunctiva under topical anesthesia. The conjunctiva will heal even if not sutured but it will take longer and the eye must be kept patched to prevent infection. It should be sutured if possible.

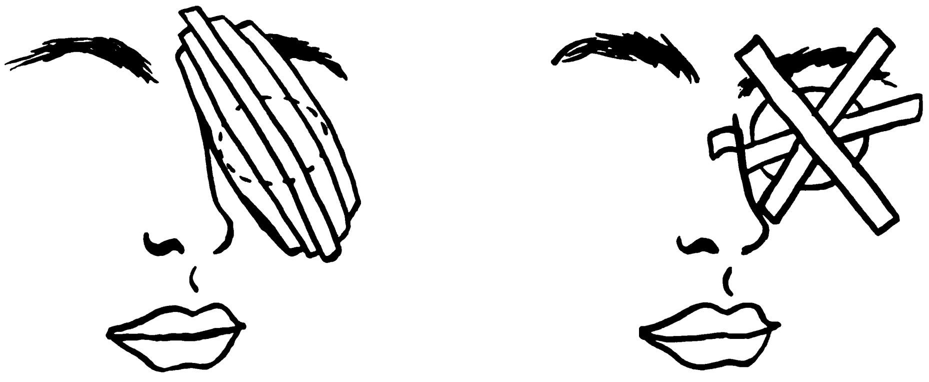
The Cornea

Abrasions.—These should be stained with fluorescein, if available, in order to determine their extent. Fluorescein is a red dye that comes in small, dry, sterile strips of filter paper. Introduced into the tears, it has the property of staining the cornea green anywhere the epithelium is missing for whatever reason. Fluorescein solution should not be used because it cannot be sterilized. In addition to staining the cornea, one should examine the eye closely (with the slit lamp if available) to make certain that there has not been a perforating injury. At least, the eye should be examined with a loupe to determine the depth of the injury as nearly as possible and to check to make certain the anterior chamber is fully formed and the iris intact (pupil round and centrally placed). If so, the proper treatment is to place a tight bandage over the eye and check it daily, keeping the eye patched until healing has occurred and there is no further staining. This may take 1 to 3 or 4 days depending on the size of the abrasion. If it is a large one and particularly, if one sees cells in the anterior chamber on slit lamp exam, the pupil should be kept dilated. This will diminish the pain and photophobia and also prevent synechia. The pain should be controlled with systemic pain relievers: codeine, Darvon, or whatever is available. Neither topical tetracaine, nor any other "caine" should be used for sustained relief of pain. It is all right to use it once or twice in order to facilitate the examination, but it should *never* be used for sustained relief. For this reason tetracaine ophthalmic ointment should never be used. Tetracaine has several adverse effects. It slows the mitoses of the cells that are trying to proliferate and cover the denuded area. It causes swelling and softening of the remaining epithelium so that it tends to slough off, particularly, if the eye is left open so that the lid continues to blink and rub off this softened epithelium. There is a rebound of pain from tetracaine after a day or two of use. When tetracaine or any related product is used to treat an abrasion, the patient comes back in a day or two with practically all of the corneal epithelium gone and he is in extreme pain. Even so, the cornea will heal with proper treatment, but it will take three times as long or longer and the patient is going to experience extreme discomfort. A tight dressing to splint the lid plus codeine or Darvon is the only treatment for abrasions.

The wrong and the right way to apply an eye dressing is shown in figure 9–40. The proper way is to place an eye pad (or gauze) over the eye and hold it in place with adhesive strips beginning near the center of the forehead and going down and laterally to the cheek, keeping as far away from the angle of the mouth as possible; otherwise, every time the patient talks, or chews, or smiles the bandage tends to work loose.

Perforating Injuries.—These can be recognized by the fact that the anterior chamber has collapsed and in some instances the iris becomes prolapsed through the wound. The iris-lens diaphragm may bulge forward due to the release of pressure from the anterior chamber and also the cornea may fall back as a result of the loss of aqueous. First aid for such an injury is: (1) Apply antibiotic drops, not ointment, since ointment must not be allowed to enter the anterior chamber; (2) patch both eyes loosely. Do not apply pressure or more ocular contents will be squeezed out. Both eyes are patched because if the fellow eye is left open, it will be looking about and the injured eye will move with it; (3) evacuate the patient, flat on his back, with sandbags to his head. The eye is now like an open cup, and if the patient lies on his side or stomach, ocular contents will drain out through the laceration; (4) tetanus booster; (5) systemic antibiotic, preferably chloramphenicol.

In suturing a lacerated cornea, some ophthalmologists reposit any prolapsed iris if the case is not over an hour or so old. It is probably best to excise the prolapsed iris in any case as this is the type of case in which sympathetic ophthalmia could possibly occur. The laceration is closed with



RIGHT WRONG

Figure 9–40. *Application of eye dressing.*

the smallest silk (6 or 7–0) with the smallest atraumatic needle available using interrupted sutures placed approximately one mm apart and with the bites going only about halfway through the cornea. One needs the best magnification possible; the operating microscope, if available, or a loupe if that is all one has. After closure the anterior chamber is reestablished by injection of an air bubble. If the air comes out at any point, more sutures are needed. The bubble will absorb in 2 to 4 days. Both eyes are kept covered with a loose bandage.

The Sclera

Lacerations of the sclera are handled in the same way as corneal lacerations. For first aid, both eyes are loosely patched and the patient is evacuated flat on his back. To suture the sclera, any prolapsed uveal tissue is excised and the wound closed with interrupted silk sutures.

Intraocular Hemorrhage

Intraocular hemorrhage can occur as a result of either sharp or blunt trauma, and is of two types.

Hyphema is a hemorrhage into the anterior chamber. What usually happens is this: Someone receives a blow to the eye, and immediately following the injury, notes that his vision is extremely blurred. On the way to the sickbay, his vision begins to clear a little. The blow caused a rupture of a small blood vessel and the aqueous filled with blood obscuring vision; then after a few minutes the blood settled out to the dependent portion of the chamber. When he gets to the sickbay, blood will be seen in the lower part of the chamber with a fluid level on top. This is the reason for the name, hyphema, meaning "blood below." A hyphema is a potentially dangerous injury. If the patient is allowed to be up and to pursue his normal activities, there is a good chance that on the second or third day, the tiny clot in the ruptured vessel will soften and loosen so that a secondary hemorrhage occurs. The secondary hemorrhage is always greater than the primary, and may completely fill the anterior chamber. Clots may form and it may become necessary to open the anterior chamber to flush them out. Further hemorrhage is possible during this procedure. Glaucoma may ensue at any point and the eye may even be lost. The tension may even be elevated soon after the injury. If high, it should be controlled with diamox. The proper treatment for a hyphema is to put the patient to bed with both eyes bandaged and keep him quiet with sedation and/or tranquilizers. A secondary hemorrhage can occur even under these circumstances but is unlikely. In most cases, when one changes the dressing every day, it will be noted that the hyphema is a little smaller each time, until it finally absorbs. Bedrest (on the back but with head and knees slightly elevated from time to time) and bilateral patching are continued for a minimum of one week or until all the hyphema has disappeared.

Vitreous hemorrhage can also result from either sharp or blunt trauma. Absorption is much slower from the vitreous and may never be complete. Patients with vitreous hemorrhage are kept on bedrest and bilateral patches for about 10 days.

Rupture of the Globe

Rupture of the globe can occur anywhere depending upon how the force is applied, but one particularly vulnerable point is at the limbus. The Canal of Schlemm makes this area weak, and blunt force applied to the anterior of the globe may cause a rupture here. When this occurs, iris is almost always extruded from the globe (but under the conjunctiva) and the pupil will "point to the injury." This type of patient should be evacuated via stretcher to the hospital. The treatment consists of making an incision through the conjunctiva 5 mm or 6 mm from the limbus, freeing up back to the injury, excising the prolapsed iris, and closing the rupture with interrupted sutures.

Chorioretinal Tears

One weak point is in the macula. Blunt force applied to the front of the globe may be transmitted back to the macula and cause a hole in the macula, or it may cause a long chorioretinal tear beginning near the disc and arching around, above, or below and going through the macula. In either case the eye has lost all central vision though peripheral vision remains. There is no treatment. Another weak point is at the ora serrata. Trauma applied to the globe may cause the retina to tear away at this point, called a "disinsertion." Vitreous can now get behind the retina and dissect it away from the choroid, producing a retinal detachment which may get larger and larger as time goes on. A reattachment procedure should be done. Any retinal detachment should be evacuated on a stretcher and if the macula is at all threatened, the patient should be kept flat on his back with bilateral eye patches and sandbags to his head to prevent movement.

Foreign Bodies

Extraocular foreign bodies (those resting on the cornea or in the conjunctival sac) are very common. Finding them is sometimes difficult if they are very small. It may help to stand first on one side of the patient and then the other and shine a light in various directions across the cornea. The patient should always fix his eyes on some object across the room so that they remain still during the examination. Instill some fluorescein into the conjunctival sac, and you may be able to locate the foreign body by the small amount of staining surrounding it. Always be sure to evert the upper lid and look in the sulcus just above the lid margin, as foreign bodies often lodge here. To remove a foreign body, instill some topical anesthetic solution; then dampen a cotton swab with the anesthetic and gently try to wipe it away. If this is unsuccessful, use a blunt spud or spatula and try to tease it away. If the foreign body does not come away easily, and if you are near a hospital, do not hesitate to refer the patient to the ophthalmologist. Although a speck in the eye may seem like a rather minor injury to send in, it is much better to do so because it is much easier to remove a foreign body under slit lamp observation. Furthermore, one can tell just how deep in the cornea one is probing with the use of the slit lamp.

Intraocular foreign bodies must be referred to the hospital, and they must be removed. Metallic foreign bodies, particularly, will dissolve, and the metal will spread throughout the eye, eventually leading to blindness. If a foreign body has penetrated the globe but is partially protruding, in most instances it is better to patch the eye and send the patient to the hospital for removal. If the foreign body has just barely penetrated into the anterior chamber and its entire extent can be seen, one would be justified in gently teasing it out, particularly if the prospect of a considerable period of time before the patient could be taken to a hospital existed. A tetanus booster is indicated in any case where there has been a penetrating injury.

Burns

Thermal Burns.—Although severe burns of the lids are common, it is rare to find a severe thermal burn of the eye itself, simply because of the protection provided by the lids, the corneal reflexes, and the tears. Usually thermal burns are superficial and can be treated in the same way as abrasions.

Chemical Burns.—These can be devastating and quick action is the only thing that will save the eye in many instances. Washing with water from any source is the best treatment regardless of the toxic substance involved. The only well-known exception to this rule concerns white phosphorous. Here mineral oil, if available, and debridement offer the only hope, and a severe injury is to be expected in any event. If the patient with a chemical burn is seen in the sickbay, normal saline is a good thing to use and no less than 2,000 cc should be allowed to flow across the eye, pulling the lids out from the globe in order to reach the fornices. At the end of the first washing, test the conjunctival sac with pH paper, and if still alkaline or acid, wash further until pH 7 is obtained. Alkali burns are worse than acid burns, depending upon the strength of the solution, of course. With alkalies, one should check the pH about two hours after the first treatment. Alkalies may dissolve into the tissues and after the washing seep back out into the tears, and cause further damage.

Radiation Burns.—*Ultraviolet* burns result from arc welding, sunlamps, and even at times from UV lights placed in hospital rooms for bacteriocidal effect. There is a lag of four to six hours from exposure to onset of symptoms. The most common cause of sudden onset of bilateral red, weeping, photophobic eyes is UV burn. Stippled staining of the interpalpebral area of the cornea is seen when fluorescein is instilled. This is a self-limiting disease and cold compresses and rest with sedatives generally seem to be the best treatment.

Eclipse blindness may occur if one stares at the sun for a matter of only 5 to 6 seconds. This permanent visual defect may result since the refracting mechanism of the eye focuses light on the macula, but it also focuses infrared radiation there. When infrared is present in sufficient quantity, the macula will be destroyed, partially or entirely, resulting in a permanent central scotoma.

In aerospace medicine, the possibility of inadvertent eclipse blindness is somewhat increased for lunar or extravehicular operations unless appropriate protection for the eyes is provided. At orbital altitudes, the intensity of solar radiation in the visible wavelengths is increased approximately 17 percent over that found at sea level (Schmidt, 1962). Although it is quite difficult to determine the exact increase in infrared energy with increasing altitude because of the absorption of this energy by variable bands of water vapor, the infrared above the earth's atmosphere may be as much as 1.5 to 2 times that at sea level.

Nuclear retinal burns are described by Matoush (1960) as a pathological condition of the eye in which there is irreversible tissue damage and some permanent loss of vision. The condition is caused by the absorption of excessive amounts of thermal energy in the retina and in underlying layers, principally the choroid.

Although nuclear explosions release energy in a broad thermal spectrum, covering the ultraviolet, visible, and infrared regions, the thermal radiation that is found to be responsible for retinal burn covers only the visible range and part of the infrared. This selectivity is the result of absorption of the other radiations (ultraviolet and/or infrared) by various elements of the eye—cornea, lens, and intraocular fluids—before they can reach the retina. After the nonattenuated radiation has succeeded in traversing the preretinal media, it is absorbed in the black pigment epithelium of the retina and in the pigment cells of the choroid. In so doing, the light energy is transformed into heat, causing a rise in tissue temperature. This temperature rise may be sufficient to cause irreversible damage (coagulation necrosis) within the absorbing tissues and within neighboring receptor elements, giving rise to a retinal and chorioretinal burn.

Susceptibility to nuclear retinal burns is traceable directly to one of the fundamental eye processes—that of optical focusing and image formation. As pointed out by Byrnes, Brown, and Cibis (1955), the radius of the image of an atomic fireball on the retina varies linearly with the radius of the fireball and the distance from the fireball. Thus, with a pupil of given size at a given distance, a certain amount of energy is distributed over the image area. If the subject is twice as far away, the amount of energy passing through the same pupil will only be one-quarter as great. However, because of the focusing power of the eye, the image area in which the energy falls will be only one-quarter as large. The energy per unit area will therefore be constant except for the attenuations produced by air and other ocular media. Thus, distance alone provides little safeguard against visual damage if one is looking directly at the explosion.

The blink reflex, occurring in 100 to 150 milliseconds, also affords little protection against nuclear retinal burn. For a one-kiloton weapon, 73 percent of the energy is delivered during the blink-reflex period. For a ten-megaton weapon, only one percent is delivered during this period. Even so, the total energy received is higher due to the tremendous increase in weapon yield.

Since the probability of looking directly at an unexpected burst at exactly the moment of detonation is quite small, there is little likelihood of retinal burn if aviators are trained not to turn their eyes toward the light source, even after the initial startle reaction has subsided. This caution is of especial importance in the case of high-yield weapons for which the highly luminous fireball may last for a number of seconds. In these circumstances, it is imperative that aviators not look toward the burst.

In their long-term effect, seriousness of retinal burns depends upon such factors as tissue involvement (size and depth of lesion) and on location in the visual field. Large burns may invite grave aftereffects such as retinal detachment; small burns are not likely to cause serious visual impairment unless they occur on the region of the retina associated with acute vision (macula) or perhaps on the blind spot (optic disc). However, of greater concern in tactical planning are the possible immediate effects of retinal burning. Descriptions of postexposure effects are meager, viz., that immediate vision loss occurs equivalent to severe flash blindness and, on recovery of adaptation, there is possible persistence of discomfort.

The above description of chorioretinal burns deals primarily with radiation levels sufficient only to produce damage to the visual system. Brown (1961) has described the various types of damages to the eyes which may result from close exposure to even higher radiation levels. In such cases, thermal damage may occur to any part of the eye. Exposure to these extreme levels of thermal radiation will result in coagulation in the corneal tissue with resulting opacities. Hemorrhagic congestion may occur in the iris. Proteins of the lens tissue may be coagulated with the development of cataracts, and there may be peeling off of superficial layers of the lens (exfoliation).

Flash blindness may occur in the event of nuclear conflict. It is probable that a pilot, in such case, will be exposed to the flash of one or more nuclear weapons during the course of his mission. Even if these occur at great distances, the energy released by such bursts is sufficient to cause retinal damage if viewed directly, or "flash blindness" if not viewed directly. This impairment of vision, even if quite brief, could cause complete failure of a mission. Inasmuch as the likelihood of experiencing retinal burn is small, as noted above, and the likelihood of flash blindness rather high due to the effectiveness of direct, reflected, and scattered radiation, the flash blindness problem is the serious one from the point of view of the pilot.

When an individual is exposed to diffuse but very intense light, his initial impression is of a very white flash which may border on the painful. For a period of up to several seconds, there is an extended blink reflex. During this time, it is impossible to open the eyes. After the eyes are opened, the colored afterimages customarily are sufficiently intense that useful vision is not regained for many seconds.

In research concerning flash blindness, it has been demonstrated that an increase in illumination of the visual task produces a substantial decrease in the period of blindness. For instance, under night cockpit-lighting conditions, the flash from a burst might produce blindness which would last for as long as 60 seconds. If the high-intensity panel lights were turned on immediately after the flash, however, this period of blindness would be reduced to from four to five seconds.

The above results lead to an obvious recommendation for coping with flash blindness. If a pilot suddenly is dazzled by the light from a nuclear burst, in all likelihood he will wish to control his aircraft by reference to his instruments, even on a day mission. He should not look out and scan the terrain since, if he should happen to focus on the fireball, he will suffer severe and permanent damage to his vision. Immediately following the flash, even though blinded, he should turn up his high-intensity panel lights. He thus will be able to read his instruments in only a few seconds and should not lose control of the aircraft.

Realizing the importance of maintaining the visual capability of Navy personnel under combat conditions, for several years the Navy has had an intensive program to develop protective devices. Devices which are available or which are under development range from gold-coated, low-transmission visors, excellent for day missions, to "active" devices which are transparent normally but which "close" when exposed to intense light. Procedures for using available devices are demonstrated during the flash blindness training program conducted at selected Aviation Physiology Training Units.

X-ray or Gamma Nuclear Radiation has adverse effects on the eye consisting of cataracts from small repeated doses, or retinal degeneration from larger doses. These effects usually result from nuclear accidents or as unavoidable side effects from radiation therapy.

Laser beams may cause retinal burns and other deleterious effects to the eye. See *Visual Effects of Lasers.*

VISION AND THE ENVIRONMENT

Color Vision

Naval aviation personnel must have color perception which is adequate for their duties. They must be able to recognize colored signals, lights, and smokes, and they must be able to discern the "meatball" for carrier landings.

Out of every 100 people, 90 have normal color vision and 10 have some color defect. Of the 10, 2 have no color perception and 8 have varying degrees of color deficiency. The Farnsworth Lantern will pass 95 out of 100 people; in other words, it will pass the 90 percent of people who are normal and the best 5 of the 10 with color vision defects. This is felt to be an adequate screen for color perception.

The Farnsworth Lantern is a black metal lantern of sturdy construction with two tiny apertures, one above the other, through which colored lights can be presented. Only three colors are used, red, green and white, and all are of the same intensity. Nine different combinations of the three colors are shown (i.e., red over green, green over white, etc.). If the subject calls all nine correctly, he passes. If he misses any combinations, he is given the test twice more and he is allowed to miss two. He must get at least 16 out of 18 correct. The combinations can be presented in different order to prevent the test from being memorized.

The color plates, either American Optical or Dvorine, can be used for screening in the field where the Farnsworth may not be available, but the Farnsworth is the final test and all candidates must pass this test at Pensacola before entering any flight program. Actually the Lantern test is easier to pass than the plates, but the plates are easier to memorize. It is possible for a person to learn to recognize the plates and call off the correct number even if he cannot see the number and even if they are presented out of order. The Lantern test can be presented in any order and cannot be memorized.

Night Vision

Physiology.—The retina is composed of two types of light-sentitive receptors. The cones, especially adapted for operation in moderate to high illumination, are concentrated in the central or foveal area of the retina. Cone, or photopic vision, produces the sensation of color and allows discrimination of fine detail.

The rods are especially adapted for operation in dim illumination. Rods are extraordinarily sensitive to small amounts of light energy and nearly attain the theoretical lower limit of sensitivity, response to one quantum of light (Ruch, 1960). Rod, or scotopic vision, cannot discriminate color and is not capable of resolving fine detail. This latter characteristic is attributed to the neural interconnection of rods through diffuse ganglion cells which allow augmentation of weak stimuli by two or more receptors. Scotopic vision is so called because under dark-adaptation, there is a central blind spot or scotoma due to the absence of rods in the macular area.

Figure 9–41 presents the basic dark-adaptation curve. The curve of figure 9–41 is seen to have two distinct parts. The first part, beginning with a rapid decrease in the threshold and leveling after about 10 minutes, reflects the adaptation period of the cone receptors. The value shown at the 10-minute period is the threshold energy for producing a cone response. The

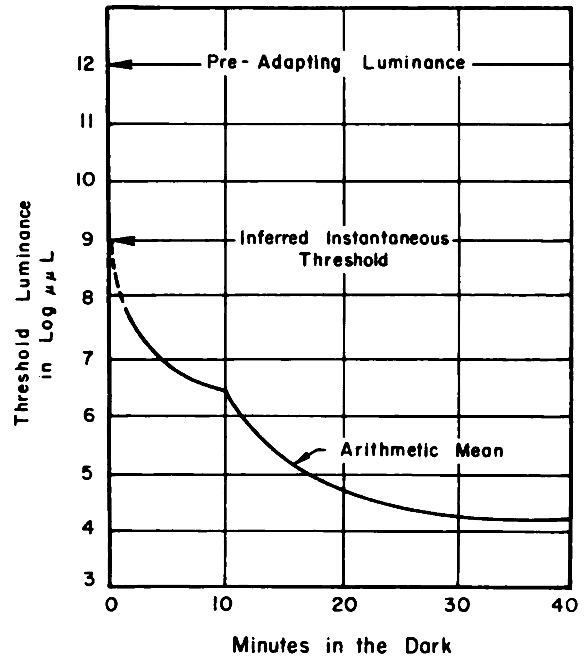


Figure 9–41. *Dark-adaptation curve* (Wulfeck, Weiz, & Raben, 1958; data from Sloan, 1947).

second segment of the curve represents the continuing adaptation of the rod elements. Upon complete adaptation, rods may respond to a range of brightness from about 0.004 mL down to 0.00001 mL (Wulfeck, Weisz, & Raben, 1958).

The retinal process whereby physical energy (light) is changed into nerve impulses involves the decomposition and regeneration of certain light-sensitive biochemicals. The primary substance involved in dark-adaptation is rhodopsin, or visual purple, which has a concentration across the retina paralleling the known density of rods (Ruch, 1960).

The exact mechanisms of adaptation are not simple. Bartlett (1965) states that, "Between the catching of the light quanta by rhodopsin and the resulting modification of impulse rhythm in optic nerve fibers, there is a complicated and highly nonlinear mechanism whose properties alter profoundly during light- and dark-adaptation." However, the essence of the process may be described in the following diagram:

$$\text{light} \rightarrow \text{rhodopsin} \rightleftharpoons \text{retinene and protein} \rightleftharpoons \text{vitamin A and protein}$$

When exposed to light, rhodopsin dissociates into protein and retinene, a pale-yellow carotenoid. These substances further break down into vitamin A and protein. For a short exposure, even though the light might be intense, much retinene and a little vitamin A are produced. Subsequent dark-adaptation is rapid because the regeneration of rhodopsin from these products is accomplished quickly. Long exposures result in a slowed adaptation curve because more retinene has gone to vitamin A and must be resynthesized by a slower route.

Detrimental Factors.—There are a number of things which reduce the ability of the rods to dark-adapt. Among them are:

1. *Exposure of the eyes to bright light.* Lying on a white beach in bright sunlight without sunglasses can significantly reduce one's ability to dark-adapt for as long as 36 hours. The same can be said for flying above white clouds with bright sun on them. Every aviator should be equipped with a good pair of sunglasses to avoid such exposure.

2. *Smoking.* All aviators should understand the effects of carbon monoxide obtained from smoking. The affinity of human hemoglobin for carbon monoxide is 200 times its affinity for oxygen. An aviator who is a heavy smoker can get as high as 10 percent carbon monoxide hemoglobin saturation. Only 3 percent is sufficient to cause measurable impairment of functions such as vision and altitude tolerance. The effects of carbon monoxide

on vision are particularly serious. Smoking three cigarettes in a relatively short period before takeoff will reduce the night vision of a pilot as much as the effect of 8,000 feet of altitude.

3. *Dietary Factors.* The most important dietary consideration for night vision is an appropriate supply of vitamin A. Aviators should know which foods contain vitamin A or carotene, and should eat them regularly. Although an adequate amount of vitamin A is necessary, an excess of vitamin A will not improve normal night vision.

4. *Hypoxia.* The efficiency of the eye depends upon the proper supply of oxygen to the retina. With a decrease in oxygen, vision is affected earlier and more severely than any of the other senses. This is particularly true for the rod system, inasmuch as the vascularization of the retinal periphery is poorer than that of the fovea. Figure 9–42 shows the loss of visual sensitivity with a decrease in the partial pressure of oxygen. Note that this decrease begins immediately upon ascent from sea level. It is for this reason that oxygen is required on all flights at night operating above 5,000 feet.

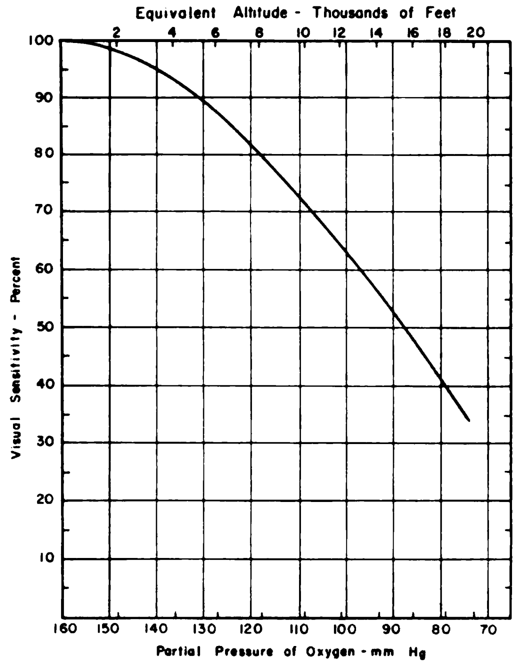


FIGURE 9–42. *The reduction of visual sensitivity (raised thresholds for light, or decreased ability to see at night) as the PO_2 in inspired air decreases* (WEBB ASSOCIATES, 1962; ADAPTED FROM MCFARLAND, 1953).

5. ***Liver disease,*** inasmuch as carotenes are converted to vitamin A here.

6. ***Certain systemic diseases*** such as sprue, which produce avitaminosis A.

7. ***Certain ocular conditions*** such as retinitis pigmentosa and choroideremia.

8. ***Acceleration.***

Red Lighting.—The use of an appropriate red lighting system will allow visual tasks to be accomplished, through use of foveal vision, while retaining the dark-adaptation of the rods. Figure 9–43 shows the operation of a red filter cutoff which allows only light at wavelengths of 620 mμ and above to be passed. It can be seen that this red light provides considerable stimulation for photopic vision while virtually none for scotopic vision. Figure 9–44 presents average dark-adaptation curves following adaptation to red light and to white light of approximately the same luminance. It can be seen that recovery from the red light is virtually complete in a matter of only several minutes.

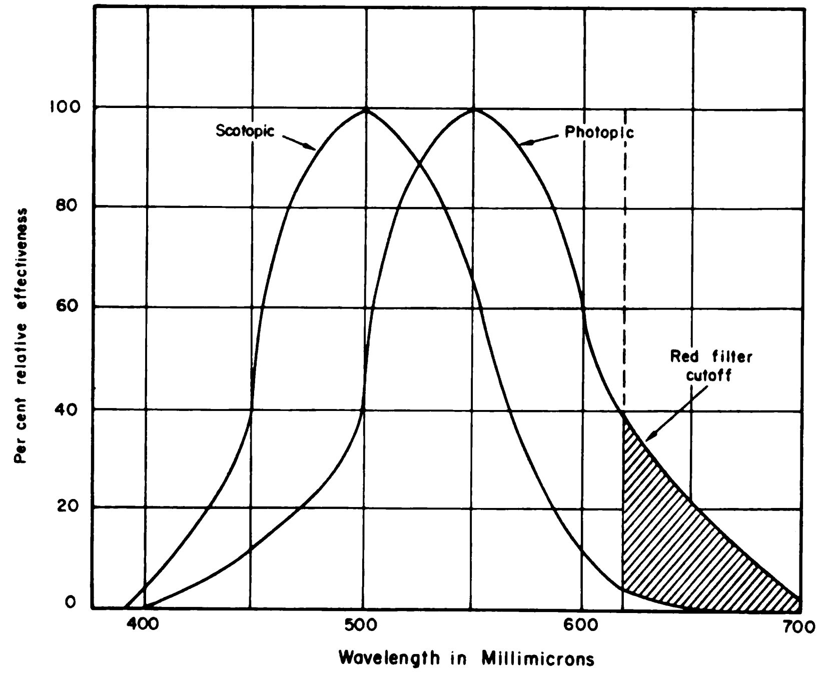


FIGURE 9–43. *Luminosity curves for scotopic (rod) and photopic (cone) vision. Since the maxima are arbitrarily set at 100, these curves give no information about the relative sensitivity of the rods and cones. The vertical line indicates the place at which a common red filter cuts off. It transmits 1/10 of the light involved in the cone curve, and 1/100 of that in the rod curve* (BARTLEY, 1951, ADAPTED FROM HECHT & HSIA, 1945).

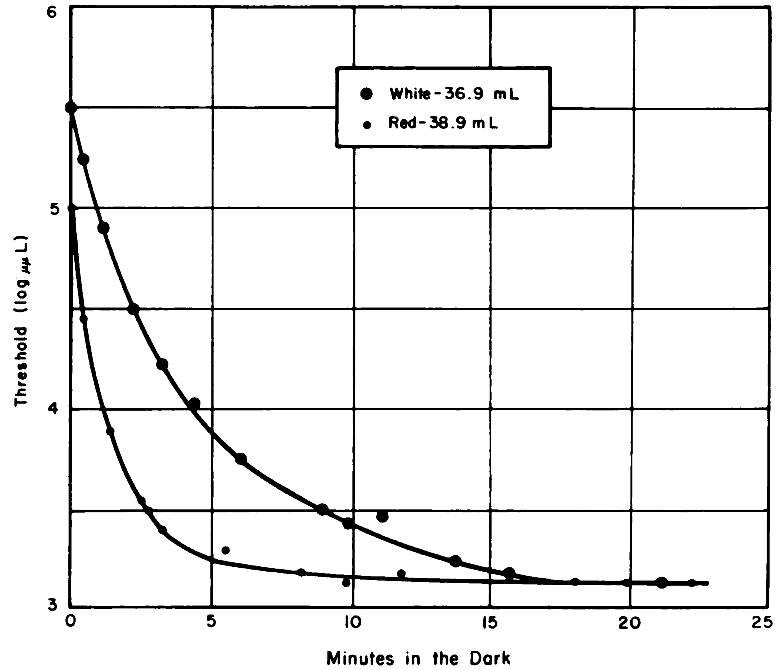


FIGURE 9–44. *Average dark-adaptation curves following adaptation to red light and to white light of approximately the same luminance* (BARTLETT, 1965, ADAPTED FROM HECHT & HSIA, 1945).

Because of the benefit in retaining the effectiveness of night vision, Navy aircraft use red lighting systems. In addition, some areas of carrier flight decks, hangar decks, and passageways leading from ready rooms to operating areas also are illuminated with red light.

There is no routine test for night vision given prior to entering any flight program. However, soon after becoming candidates for a flight program, all students are given a course of instruction by the Aviation Physiological Training Unit which includes a Night Vision Trainer. Should any student give an indication that he does not see the silhouettes as presented, he is tested on the *Radium Plaque Adaptometer*. This device consists of a black bakelite case that is kept closed at all times except in complete darkness. Inside there is radioactive background that gives off a very faint glow. Within the glowing background there is a letter "C" which is perfectly round except for the opening of the C. The device is held in all four positions and the examinee is asked to tell whether the open part of the C is right, left, up, or down. The test is performed at different distances and the farthest distance at which the examinee can call all four positions correctly is determined. If the adaptometer must be held less than 5 feet from him in order for him to be able to call all four positions correctly, it is considered unacceptable, from 5 to 7 feet is below average, 7 to 9 feet is average, and 9 to 11 feet is above average.

The Night Vision Trainer.—The Night Vision Trainer is a device to teach pilots and student pilots how to utilize their night vision. There are night vision trainers located at Aviation Physiological Training Units (listed in chapter 4) throughout the country. In such trainers, the class is brought in from ordinary daylight to what appears to be a room of complete darkness. There is, however, at the time the students enter the room, a group of landscape silhouettes cast on the front wall of the lecture room by means of a very dim light.

While the students sit in apparent complete darkness, the instructor explains the physiology of the retina. He calls the attention of the students to the fact that the macula, which contains no rod elements, is completely blind to dim light. As the students listen, the rod elements which were inactive when they entered the dark room, gradually regain their sensitivity. The instructor calls for any students who sees any object during the demonstration to speak up.

The instructor now suggests that students will see more clearly in dim light if they will look, not directly at the object of regard, but slightly off to the side. This, the students find, does work. They find that by scanning the simulated horizons before them they can pick up smaller and more difficult silhouettes with their off-center or peripheral vision.

Each student is now asked to close one eye tightly and to cover it with his hand. The full lights are switched on for about one minute. The silhouettes disappear. The full lights are switched off again and the student sees for himself that the eye which was covered is able to see the silhouettes as before but the eye exposed to the bright light is blind. Therefore, the flyer is advised that if he must use white light to read a map at night he must do so with one eye only; then at least one eye will remain dark-adapted.

The students now put on red goggles. The lights are switched on for a minute. They are told to keep their eyes open. Again the lights are switched off and the red goggles removed. The student finds by actual experience that a red light will not disturb his night vision.

The instructor now sums up the lesson. He advises student pilots that to obtain night vision, they must dark-adapt their eyes by remaining in complete darkness for about 30 minutes. This period can be shortened to 5 minutes if red lenses have been worn for at least 25 minutes before entering complete darkness. Once dark-adapted, the flyer must not allow his eyes to be exposed to any but very dim white light. Any reading must be done in red light. Incidentally, he is advised not to try to read red print in a red light for he cannot see it. This vivid practical experience convinces the future pilot that there is a right way and wrong way to prepare for night flying. However, experience has proven that the Flight Surgeon should frequently brief his squadron on this subject to refresh their memories—especially when night flying is performed infrequently.

Special Problems

Night Myopia.—Everyone has a slight "myopic shift" under conditions of dark-adaptation. That is, each of us is either more myopic or less hyperopic when dark-adapted. This is due to what is known as the "Purkinje shift." Under conditions of light-adaptation, the retina is most sensitive to orange and red hues, but when dark-adapted, the greatest sensitivity shifts to the blue. Blue lights can be seen more easily at night, and incidentally, this is the reason that many police departments have changed the flashing lights on their patrol cars from red to blue. However, even white light, when it enters the refractive mechanism of the eye, is split into its various components. There is never an exact point focus but rather a slightly elongated focus in which the red rays come to a focus farthest back in the eye and the violet farthest forward, with the other colors of the spectrum in between. The distance between the red focus and the violet is extremely small, yet it is sufficient to be significant. Actually, it doesn't take much distance to be significant. 1 mm. in actual length is equivalent to 3 diopters of power, so it only takes $\frac{1}{12}$ mm in length to equal 0.25D, and, as mentioned earlier, 0.25D of spherical power can, under certain circumstances, make a difference of one line on the eye chart. Thus it can be seen that $\frac{1}{12}$ mm can be significant. Under conditions of dark-adaptation, the accommodative mechanism of the eye tries to focus the blue rays on the retina because these are the ones the eye sees best. If one is hyperopic, he can merely relax his accommodation the proper amount and have no difficulty. However, the emmetropic or myopic eye cannot do this, so the emmetropic eye becomes slightly myopic and the myopic eye becomes more myopic. Anyone who has performed a number of clinical refractions has noted that when a myope comes in for a change of glasses, it is quite often because he has begun to have trouble reading road signs at night. The situation can become critical for an aviator, if he is emmotropic, and even more so, if he is mildly myopic. He might be —0.25 myopic, for example, and still be able to read 20/20 in good light. At night, however, he may be "effectively" —0.50 or even —0.75 myopic because his retina is trying to see the images formed by the blue rays. If a small degree of tension is added to this (and who is not a little tense when making a night carrier landing for instance!), he may have a little ciliary spasm which might make him "effectively" as much as —1.00 or —1.25, which is, as the Flight Surgeon knows, equivalent to 20/50 or 20/60 vision. It is known that a number of pilots who reported difficulty in seeing the "meatball" at night were all found to have refractive error under a cycloplegic in the range of PL to —0.50. None were hyperopic. A few of these pilots obtained myopic corrections of —0.25 or —0.50 sph and made landings with them. They reported that they had no difficulty picking up the "meatball' and were able to make better than average landings with them. At present, however, the practice of wearing lenses for such landings is not authorized.

Space Myopia or Empty-Field Myopia.—In the normal everyday environment, there is something to look at all the time and the accommodative mechanism constantly keeps the eyes focused at various distances, depending upon the location of the object of regard. At altitude, however, there may be nothing within view for the eyes to fix on unless it be within the cockpit or perhaps marks or foreign material on the canopy. Under such conditions, the eyes tend to overaccommodate, even when attempting to look far out in the distance, as when trying to sight other aircraft. This overaccommodation may be as much as 1 or 1.5D, so that effectively the eyes are —1.00D to —1.50D myopic. It is possible to miss seeing other aircraft for just this reason. To avoid this, one should look at the ground from time to time or at a distant cloud, or even at a wingtip, if it is as much as 20 feet away. On cross-country formation flights, the leader should be changed from time to time. The wingmen are looking at the aircraft ahead of them in formation and are unlikely to develop space myopia, but the leader, having nothing to look at, may do so.

Target Detection with Photopic Vision.—In the aviator with 20/20 vision, detection of a target is dependent on several factors, among which are the following:

1. Target size and distance (relative target size).
2. Overall brightness (luminance).

3. Brightness and color contrast between target and background.
4. Location of eye focus.
5. Angle between central visual axis and target.

There is a threshold of relative size which will barely stimulate the retina. The units of relative size in this context are minutes of visual angle or minutes of arc subtended on the retina by the target.

If an object (target) of threshold relative size is present, overall brightness and brightness contrast are the most important determinants of target detection (excepting motion).

Brightness is a function of luminance of an object. A luminance measurement unit is the foot-Lambert. The amount of light falling on an object (its illuminance) in foot-candles, multiplied by the reflectivity of the object (in percent), yields the luminance of the object in foot-Lamberts. Luminance values for a number of visual stimuli are presented in figure 9–45. The luminance values shown range from the minimum luminance that can be detected under the most favorable viewing conditions to that which will produce permanent damage to the eye if viewed for more than a very brief period.

	Scale of luminance (millilamberts) ↓	
	10^{10}	
Sun's surface at noon	10^{9}	Damaging
	10^{8}	
	10^{7}	Photopic
Tungsten filament	10^{6}	
	10^{5}	
White paper in sunlight	10^{4}	
	10^{3}	
	10^{2}	
Comfortable reading	10	
	1	Mixed
	10^{-1}	Scotopic
White paper in moonlight	10^{-2}	
	10^{-3}	
White paper in starlight	10^{-4}	
	10^{-5}	
Absolute RL	10^{-6}	

FIGURE 9–45. *Luminance values for typical visual stimuli* (RIGGS, 1965).

Brightness contrast is computed by $\frac{\Delta B}{B} \times 100$ where ΔB is the difference in luminance between the object and the background, and B is the luminance of the background.

Figure 9–46 relates object size, background luminance, brightness contrast, and type of retinal receptor stimulated.

Location of eye focus has been discussed previously, under the heading of empty-field myopia.

Visual acuity reduces rapidly as the image moves away from the center of the visual field, as shown in figure 9–47 (Sands, 1967). A target will be detected at maximum distance only if the observer happens to look within a degree or so of its position. An approaching aircraft which is farther away from the detector pilot's central visual axis must be nearer to him to be detected. For example, if an aircraft which could be detected at 7 miles approaching from the center of the detector pilot's visual axis were to approach instead at an angle of 10° from that central visual axis, it could not be detected until 0.7 mile away.

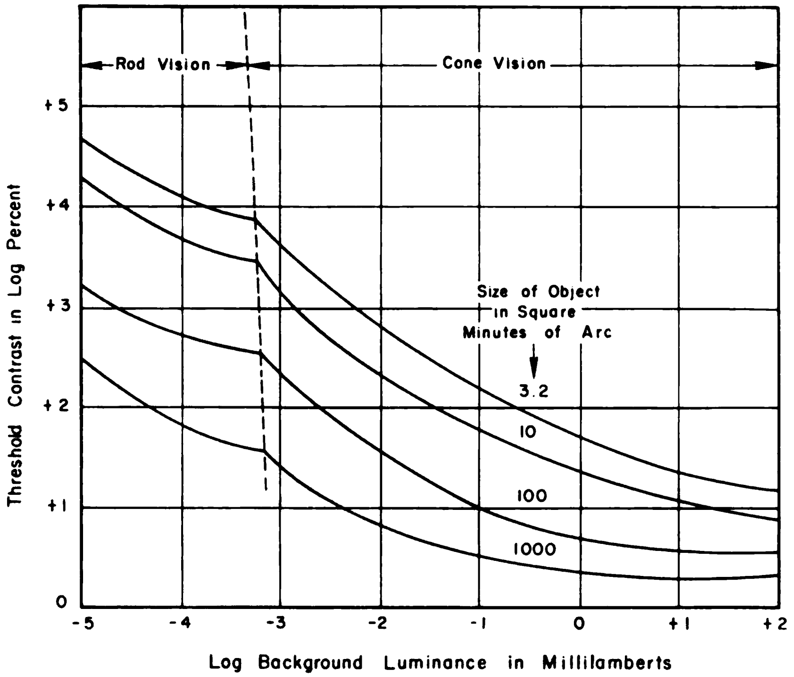


FIGURE 9–46. *Contrast discrimination curve—the smallest brightness contrast that can be seen, as a function of background luminance. Curves for test objects of four sizes are shown* (WULFECK ET AL., 1958; DATA FROM BLACKWELL).

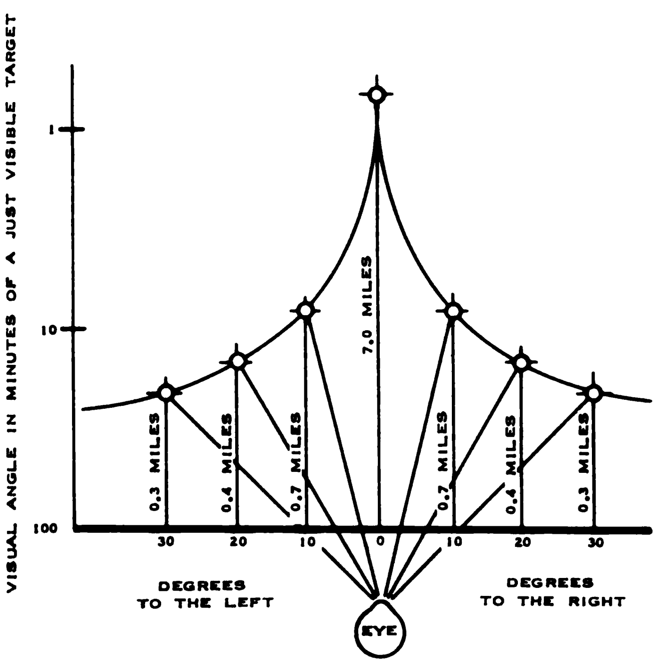


FIGURE 9-47. *The effect of offset viewing angle on the detection distance for fighter-size aircraft. Note minutes/mile ratio* (GRETHER, 1958).

VISUAL ASPECTS OF AVIATION SAFETY

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EYE PROTECTION

Aircrew

Equipment furnished to the aviator to protect his eyes and his visual capabilities includes a visor mounted on the crash helmet, sunglasses, and special types of goggles when needed.

The visor protects the eyes against windblast on ejection or jetblast while on the deck. It protects against foreign bodies entering the cockpit on the ground while the canopy is open. These may have a considerable velocity imparted by jetblast or prop/rotorwash. It protects the eyes and face against flashburn in the event of fire, and against secondary missiles in the cockpit when hit by enemy fire or birds. Finally, it also serves to protect from glare by reducing light transmission.

There are problems associated with use of the visor due to its dual functions. If the main function of the visor is considered to be eye protection in the eventualities noted above, and the secondary function protection against glare, then the visor selected should be a high-light-transmission, neutral-grey type. If the priorities are the other way around, then the low light-transmission green-tinted type should be selected. Present helmets offer the use of only one visor at a time, but a field kit is under evaluation which would alter all helmets to a two-visor system. This system has some disadvantages, however, including an increase in total weight and in mass-distribution changes.

The reason for selection of the neutral-grey visor when eye protection is paramount is that takeoffs and landings at night or under conditions of reduced visibility require it, if the visor is to be used at all. Glare protection at altitude must then be provided by the excellent sunglasses provided to each aviator, worn in addition to the visor.

The sunglasses have been especially designed and manufactured to:

1. Be free of refractive error.
2. Avoid interference with peripheral vision.
3. Avoid interference with communications (i.e., the temples are flat and fit comfortably under the helmet earcup).
4. Give light transmission of about 15 percent, which is sufficient for adequate vision in sunlight.

The color of sunglasses is not too important, but a green tint is preferred because the retina is slightly more sensitive to the green band of the visual spectrum. Disadvantages of sunglasses are:

1. Color perception is slightly impaired.
2. Visual acuity is diminished in low illumination conditions due to decreased light transmission.
3. Since visual acuity is diminished, depth perception is also decreased.
4. In some aviators, there is some minor constriction of peripheral vision by the frames.

Logical recommendations for the aviator who uses only a single visor include selection and mounting of the neutral-grey high light-transmission visor, which he would utilize throughout the flight. When necessary to combat glare, he would utilize the sunglasses. This does not appear to be the aviator's choice at present. The aviator mounts the green-tinted visor and does not use his sunglasses under the visor. He does utilize the sunglasses in glare situations on the ground, thus preventing discomfort and preserving night vision adaptability.

Groundcrew

Eye protection in groundcrew, especially on carrier decks, is important. One of the most common eye injuries encountered in groundcrewmembers is caused by metallic foreign bodies imbedded in the cornea by jetblast or prop/rotorwash. The prevention of such serious injuries is a matter of concern to the Flight Surgeon, the Flight Deck Officer, and all personnel involved. Enforcement of rules for flight deck safety, such as the wearing of goggles, is not the Flight Surgeon's job. If, however, he observes breaches of these rules, he should inform the appropriate line officers so that remedial action can be taken before injuries occur.

ACCIDENT AND INJURY AVOIDANCE

Visual Acuity

A crewmember with a large refractive error should only be allowed duty on a flight deck or on the flightline ashore if fully corrected by lenses. Such a man should always have a duplicate pair of spectacles while aboard ship or at a field activity, because the accidental loss rate of spectacles during deployments is rather high and, without them, he is a hazard to himself and others. If he should lose his last pair of spectacles, he should be removed from duty on the flight deck or flightline until replacements can be procured.

Limited Peripheral Vision

A groundcrewmember whose peripheral vision is limited because of improper wearing of goggles (or because of anything else) may suffer serious injury on the flight deck or flightline. He may not see a jet turn its tailpipe toward him, and thus be caught by the jetblast. He may not see the propeller or rotor blade that he steps into. He may cause "crunches" (minor collisions of aircraft or equipment) on the flight deck, hangar deck, or flightline because of his diminished peripheral vision.

The Naval or Marine Corps aviator simply will not accept an item of flight equipment which restricts his visual field. He depends on peripheral vision to a marked degree during flight, both for safety and for successful mission completion, and his attitude is certainly understandable.

Color Vision

Reference to the *Manual of the Medical Department* will show that color vision standards are nonexistent for many naval enlisted personnel rates. Yet these personnel are largely responsible for the everyday operation of the aviation establishment. The Flight Surgeon should remain aware that some of the jobs performed by enlisted personnel require normal color vision, despite the absence of a required standard. When such a job is noted by the Flight Surgeon, he should take official action to see that the cognizant line officer is so informed, and to insure that no unqualified person is allowed to perform such duties.

A classic example occurred when both catapults were fired simultaneously on an aircraft carrier, thereby causing an aircraft accident. In the ensuing investigation it was determined that the signal to fire the port catapult was to be given by the catapult officer with a red-lighted wand while the signal to fire the starboard one was to be given by an identical motion of a green-lighted wand. Therefore, the two catapult operators were dependent on color vision to determine which catapult to fire. The job qualification for operating the catapult was simply to be rated as an aviation boatswain's mate and the physical standards for that rate did not require testing of color vision. The investigation showed that the accident in question was due to other causes, and examination of the catapult operators showed that both had normal color vision. The fact remains that it was theoretically and actually possible to have two color-blind catapult operators whose signal to fire would require color perception.

As noted earlier, the color plates can be memorized so that a color vision test can be passed by a person with defective color vision, by depending on cues other than color. For example a veteran pilot of eight years experience in carrier flying was found to have defective color vision. When asked how he could tell whether a traffic light ashore was red or green, he stated that he waited to see if other traffic moved through the light. He also memorized the position of the red light so that when he saw a light in that position he stopped. This aviator might do very well until the day when the only cue available to him in a hazardous situation happened to be a color cue.

Midair Collisions

Causes of midair collision are many and interrelated. Improper flying technique, disregard for rules and regulations, carelessness, crowded airspace, illusions of various kinds, and darkness all may be factors. This section focuses on those collisions which occur when visibility of other aircraft is impaired by large incidence angle and high speed. Such collisions constitute a considerable proportion of the total.

Present-day Navy and Air Force fighter aircraft have supersonic speed capability. Shortly, commercial airliners also will be flying at these speeds. With increased speed, the ability to detect other aircraft visually decreases. Although commercial airlines operate under rather close radar surveillance, midair collisions still occur on infrequent occasions. Military pilots, who frequently operate outside the radar network, run an even greater risk of midair collision. The ability to detect other aircraft visually remains important.

In table 9–1, time and distances are listed for a maximum closing speed of 1,200 mph. (One should remember that the maximum closing speed of F–4C aircraft on a head-on collision course would be in excess of 2,800 mph.) At these speeds, sighting another aircraft one or two miles away on a head-on collision course can result in collision before either pilot can prevent it. Closing rates are too great to allow for necessary decisionmaking and response times. Electronic collision-avoidance systems are in the development and evaluation stage and may be in use very soon, but adoption by airlines may not be accompanied by adoption for military use.

Delays in the perception of other aircraft can be minimized by the establishment of a systematic scan pattern to cover the area in which other aircraft are most likely to appear. The optimum distance for side scan depends upon speed. At high speed, greatest danger is in the narrow cone directly ahead of the aircraft. At slower speeds, the danger area extends farther to either side and may even include the rear.

Good air discipline can contribute significantly to collision avoidance. In aircraft carrying two pilots, in-cockpit procedures should be established so that one member is continuously scanning for other aircraft.

TABLE 9–1

Time Intervals Required Between First Sighting of Object and Changing Flightpath to Avoid, and Distances Traveled in These Intervals

Operation	Time in seconds		Distance traveled in feet			
			Closing at 600 mph on stationary object		Two aircraft closing at 1,200 mph	
	For operation	From 1st sighting	During operation	From 1st sighting	During operation	From 1st sighting
Perception:						
Sensation (light travels from retina to brain)	0.10'	0.10	88	88	176	176
Motor reaction to prearrange eye movement	.175	.275	154	252	308	484
Eye movement	.05	.325	44	286	88	572
Focusing with fovea	.07	.395	62	348	124	696
Perception (minimum recognition)	.65	1.045	572	920	1,144	1,840
Decision:						
Deciding what to do (estimated minimum)	2.0	3.045	1,760	2,680	3,520	5,360
Response:						
Operating controls	.40	3.445	352	3,032	704	6,064
Aircraft changes flightpath	2.0	5.445	1,760	4,792	3,520	9,584

Visual Effects of Lasers

Many military applications for laser technology are under consideration, varying from distance-ranging devices to communications systems. Lasers can cause serious injury to the eye and the Flight Surgeon will be called upon to recommend appropriate safety practices in the field, in the shop, in the aircraft, and perhaps even in the laboratory.

Kaufman (1966) notes that laser light can damage the eye in two ways: (a) intense visible light may damage the retina, and (b) ultraviolet and infrared may damage the retina or be absorbed by the cornea, lens, or vitreous.

Retinal burns have been observed in two cases where individuals worked with an instrument having a 0.2 joule output (Jacobson & McLean, 1965).

The area of the visual field in which the reflection had been seen corresponded to the area of the retinal lesion, and the visual field loss corresponded in size and location to one of the lesions.

Kaufman gives the limits of safe exposure to laser light in his April 1966 *MicroWaves* article, "Protect Your Sight From Laser Light!" (table 9–2). In order to utilize this table, simply take it to the person in charge of the laser-using activity and ask him if his laser exceeds these limits. If it does, a problem exists and suitable measures must be undertaken.

TABLE 9–2. *Limits of Safe Exposure to Laser Light*

Maximum Safe Levels

Laser operation	Power density incident on retina	Equivalent input to the Eye
CW ($t_p > 0.1$ sec.)	0.35 W/sq cm	2.7×10^{-7} W.
Normal pulsed $t_p \approx 500\ \mu$ sec	3.2×10^{-2} J/sq cm	2.5×10^{-8} J.
Q-switched pulsed $t_p \approx 30$ n sec	2.7×10^{-3} J/sq cm	2.1×10^{-9} J.

NOTE: This table is based in part on measurements with ruby and Nd lasers. For extrapolation to other wavelengths, the data of figure 9–48 (9A vs λ) should be included since tissue absorptance influences the degree of photocoagulation and consequent damage. These threshold values should be considered accurate only to within an order-of-magnitude tolerance. Greater accuracy is precluded by case-to-case variations in the following key parameters:

1. *Tissue*

- *Pigmentation:* Varies with the individual and location within the eye.
- *Spectral absorption:* Maximal for wavelengths of about 400 to 550 mμ; diminishes toward the infrared; not much known about the ultraviolet.
- *Blood circulation:* Available blood-flow acts as stabilizing mechanism tending to maintain tissue temperature.

2. *Eye*

- *Pupil (iris) size:* Variable aperture controlling the fraction of incident light to be admitted (assuming its diameter is less than that of the incident beam); may also affect retinal image size.
- *Spectral transmission:* Very high from 400 to 900 mμ and high at several infrared bands. (See figure 9–48.)
- *Convergence* power of the cornea and lens: Affects the location of the focal plane and retinal image size.

3. *Environment*

- *Laser parameters:* Power, pulse duration, and rate, wavelength, beam divergence.
- *Location:* Eye in near or far field of source's effective aperture; interposition of lenses between laser and eye; scattering properties of targets.
- *Spatial homogeneity:* Imperfections in laser crystals; multimolding; atmospheric anomalies.

Kaufman gives the following safety rules for laser operation:

1. *Never* look directly into a laser beam. Most lasers—both cw and pulsed—can seriously damage the eye at short range. Moreover, because of

the slow spreading of the beam with distance, this danger is not just restricted to short ranges. A curious bystander, relatively far from a source, could still receive burns of the eyes and skin.

2. *Never* use the eye to detect a laser beam, and never aim an energized laser with the eye. Because of many variables, no absolutely safe limit has yet been set for eye exposure to a laser beam. Consequently "safe limits" (table 9–2) should be used only as guidelines; exposure "as close to zero as practical" should be the goal.

3. Don't rely on any safety device—for example, goggles—for viewing the laser beam *directly*. Laser goggles should be used only when they give at least a tenfold protective margin over the "safe limits."

4. Take special care to prevent reflections from watch crystals, watch bands, rings, tools, jewelry, doorknobs, glass, etc. Such reflections are difficult to predict and can make off-axis viewing just as dangerous as on-axis viewing.

5. Guard or shield all laser setups, so that personnel—particularly transient, unknowing individuals—cannot be injured by accidental exposure to the laser beam.

6. Post warning signs at the entrance to the laser laboratory. A commonly used device is a bright, flashing red light over a door indicating that a laser experiment is in progress.

7. Within the laser laboratory, use some audible/visible signal to indicate that a laser is operating or about to be triggered. Signals should be so set up that they attract attention from, rather than toward, the laser apparatus. Signs warning all persons that eye hazards exist within the room should be posted prominently.

8. Operate lasers in well-lighted rooms whenever possible. Enlargement of the eye pupils occurs in darkened surroundings, exposing the retina to greater danger in the case of a mishap.

9. In laser demonstrations, use screens with diffusely reflecting surfaces, such as white matte. If a semidiffuse or glossy target is required, its reflectivity should be such that the tentative "safe limits" are not exceeded. Precautions should always be taken to prevent any type of specular reflection from reaching the unprotected eye.

10. For manually triggered pulsed lasers, count down, look away, and close your eyes just before turnon. Forewarn all observers if repetitive pulsed operation is to follow. Take steps to prevent accidental energizing of pulsed

devices. Discharge capacitors and turn power off before making any adjustments that require placing one's body in the beam's path.

11. Beware also of high-voltage energy sources; vaporization or fragmentation of toxic materials; sources of intense light (for crystal pumping) such as exploding-wire light sources; gas and liquified coolants; fragmenting high-speed rotary equipment (for example, rotating mirrors in Q-switching experiments or rotating chopping discs); and high levels of visible and ultraviolet radiation with resultant production of ozone.

12. Establish an eye-checkup program for all exposed personnel. Ophthalmologic examinations should be given at regular intervals and should include a fundus examination of both eyes, to determine whether any retinal scarring has resulted from unknowing exposure to a laser beam. New personnel, especially, should be examined, regardless of date of the next regularly scheduled periodic examination. *Report immediately any accident in which there is the slightest suspicion of possible eye damage.*

13. Because of potential shock hazards, especially from high-energy capacitor banks, see that operating personnel are familiar with artificial respiration techniques or the location of the nearest first aid specialist at the site. At least two persons should be present at all times when lasers are operating.

14. Inform all personnel working nearby of the potential hazards involved. Stress particularly the extraordinary danger of eye damage due to the eye's high optical amplification and absorption.

Protective Devices. The industrial hygienist should have complete information on available protective eye shields or goggles. However, in many field activities of the Navy and Marine Corps, including aircraft carriers, no industrial hygienist will be available Therefore, the Flight Surgeon must be prepared to advise the line commander on needed protective devices and to consult with maintenance personnel regarding this important aspect of sight conservation.

Figure 9-48 from Kaufman's article shows the vulnerability of the retina to injury.

Figure 9-49 shows the optical density versus wavelength of commercially available protective lenses along with the wavelengths of commonly used lasers. To utilize this curve, determine the laser wavelength of the laser being utilized, and then select the protective lens which has its lowest transmittance (or highest optical density) at this wavelength. *Again, never depend on any protective lens for direct viewing of the laser beam.*

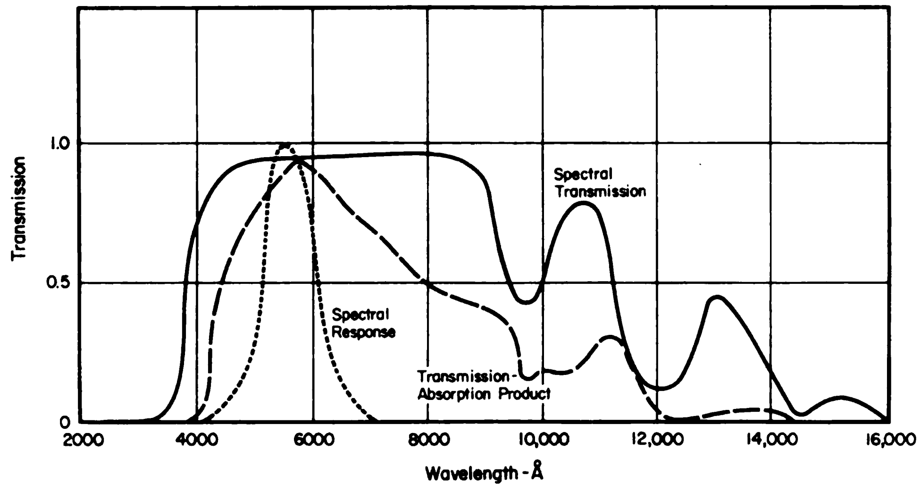

FIGURE 9–48. *The spectral transmission of the ocular media of the human eye is indicated by the black curve. The spectral response of the human eye—the so-called luminosity function—is shown by the dotted line. Actual vulnerability of the retina is indicated by the broken line—the product of the eye's transmission and the retina's absorption* (KAUFMAN, 1966).

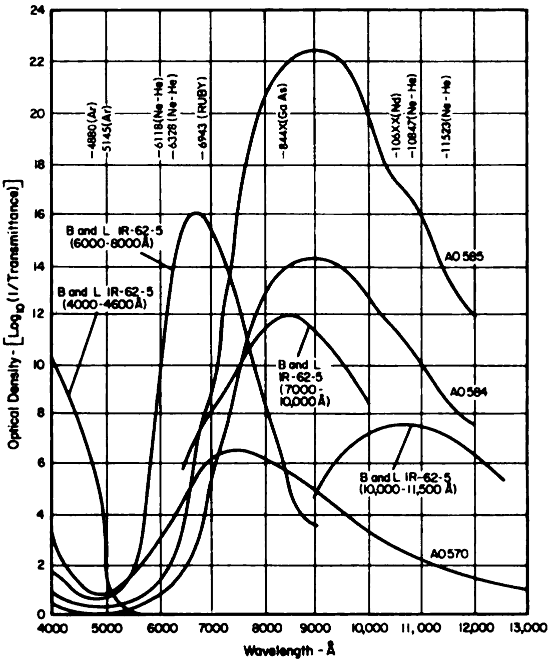

FIGURE 9–49. *Eyeshields are compared. Optical density versus wavelength is plotted for several commercially available protective eye goggles. The wavelengths of several common lasers are indicated at the top* (KAUFMAN, 1966).

For further information concerning safety equipment, Kaufman's article should be read in its entirety. Due to the relative newness of this field, a separate bibliography from his article is given below and should be referred to for more detailed information.

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Combined Stresses

The Flight Surgeon can contribute to flight safety by impressing upon pilots the cumulative effects of independent and seemingly insignificant stresses. For example, an aviator with a mild headcold, who spends an afternoon on a sunlit beach and consumes even a small amount of alcohol, may substantially increase the probability of collision during a subsequent night mission as a result of reduced night vision capability or greater susceptibility to disorientation. Obviously, no definitive data are available on the total detrimental effect of such factors acting simultaneously upon a pilot. It is, however, the duty of the Flight Surgeon to inform pilots of the facts known about each such variable and means of minimizing the effects of each.

VISUAL ILLUSIONS

Autokinetic Illusion

An individual in virtually total darkness, observing a fixed point-source of light, will report seeing the light move. Individuals also have reported such movement when viewing a stationary black target against a homogeneously illuminated visual field. Such apparent movement of a fixed spot of light is known as the autokinetic illusion. In the appropriate set of circumstances, most people will experience such movement. The autokinetic effect can, and does, produce very dangerous situations during night flying. The following report illustrates the manner in which this effect operates (Clark & Nicholson, 1953):

"While in a night bounce pattern, I followed a group of lights upwind, thinking it was another plane. Not until I was almost over it did I realize it was a lighted oil well. It seemed as though it was moving, but in no particular direction."

The autokinetic illusion can be attributed mainly to the involuntary movement of the muscles that control the eye. Under normal conditions, the perception of apparent motion of an object is controlled by other objects in the visual field. During night flying, the visual field is impoverished, and small light sources appear to move of their own accord. Inasmuch as training is ineffective against the autokinetic illusion, aviators must be trained as to its operation and must learn to deal with it as it occurs.

The *oculogravic* and *oculogyral* illusions have been covered in chapter 8. Though they both involve apparent movements of visual targets, they are due to the vestibular mechanism.

The Oculo-Agravic Illusion

The oculo-agravic illusion (Gerathewohl & Stallings, 1958) has been demonstrated in aircraft in conditions which produce brief periods of reduced or zero-gravity forces. As an aircraft enters a zero-G parabolic maneuver, a fixed visual target will appear to rise. When the aircraft reaches the zero-gravity phase, the target moves downward, with a subsequent rise again during the recovery pullout.

UNUSUAL RESPONSES TO VISUAL STIMULATION

Flicker Vertigo

An unusual pilot response to a steady light flicker is occasionally encountered. Despite its rarity, the Flight Surgeon and all pilots should be aware of it and of its devastating effects. A steady light flicker, at a frequency between approximately 4 to 20 per second, can produce unpleasant and dangerous reactions in normal subjects, including convulsion, nausea, unconsciousness, or vertigo. The exact physiological mechanisms underlying such reactions are not known. However, it is believed that susceptibility is increased when the pilot is fatigued, frustrated, or in a state of mild hypoxia. The following is a dramatic report (*Approach,* 1956) of the manner in which flicker vertigo can occur:

> After flying for some time at an altitude of 16,400 feet, a pilot in a single-seater propeller aircraft made a perfect landing. However, he did not taxi

> the plane to the hangar. Instead, the plane remained motionless, its propeller revolving slowly. The pilot was found bent over the controls, unconscious.
>
> At first it looked as though the pilot had not used his oxygen mask. However, in this case, the pilot had lapsed into unconsciousness after making a good landing.
>
> The rays of the low-lying sun were shining on the slowly turning propeller blades. Reflected flashes of light were being thrown on the pilot's face at a rhythmic rate of about 12 per second.

Johnson (1963), in a study of 102 Navy helicopter pilots, attempted (1) to determine the incidence of flicker vertigo or flicker problems during actual flight operations and (2) to determine if any helicopter pilots in an operating squadron would reveal undue sensitivity to light as shown by marked EEG changes or unusual subjective sensations during exposure to photic stimulation in the laboratory. One-fourth of the pilots reported flicker during flight as annoying or distracting but in only one instance was a near-accident attributed to flicker. None of the EEG responses of this group to photic stimulation could be classified as even borderline abnormal. Photic stimulation thus does not appear to be a useful device to detect those who would show abnormal EEG activity during flicker. Photic stimulation, however, did identify pilots who had subjective feelings of discomfort during the flickering light. In addition, and perhaps of considerable importance, photic stimulation identified 22 pilots who became drowsy and showed lowered alertness during the period of stimulation.

Pilot Fascination

Fascination is defined as a condition in which the pilot fails to respond adequately to a clearly defined stimulus situation in spite of the fact that all of the necessary cues are present and the proper response available to him. An analysis of pilot experiences with fascination (Clark, Nicholson, & Graybiel, 1953) classified these experiences into two categories:

Type A fascination is fundamentally perceptual in nature. The individual concentrates on one aspect of the total situation to such a degree that he rejects other factors in his perceptual field. "Target" fascination is of this type and has been a fairly frequent cause of aircraft accident. The pilot becomes so intent on hitting the target on an air-air gunnery run that he fails to observe the tow cable and collides with it. On an air-ground mission, he may become so intent on getting his bomb on target that he fails to observe his altimeter and pulls up from his dive too low. The following is another example of Type A fascination:

> My instructor was teaching me how to make emergency landings on a small field. I had made one or two tries and hadn't been very successful. The next time I was determined to make a good approach. Both the instructor and I were so completely engrossed in the task that we failed to hear the landing gear warning horn. Consequently we landed with the wheels in the up position.

In Type B fascination, the individual may perceive all of the significant aspects of the total situation, but still be unwilling or unable to make the proper response. The following is an example of Type B fascination:

> I went into a skidded turn stall during a small-field shot. I knew I was in unbalanced flight during the last turn, but as I recall, I was so determined to get a straightaway before hitting the field that I didn't seem to care what happened. The plane stalled and the instructor took over.

Fascination has apparently been experienced by virtually all military pilots. The aviator must be made aware of such hazards and periodically reminded of them.

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CHAPTER 10

The Cardiovascular System in Aerospace Medicine

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CARDIOVASCULAR PHYSIOLOGY
- Positive Acceleration
- Negative Acceleration
- Transverse Acceleration
- Weightlessness
- Hypoxia
- Reduced Atmospheric Pressure

CLINICAL EVALUATION OF THE AVIATOR
- Medical History
- Physical Examination
- Radiology
- Electrocardiography
- Other Diagnostic Procedures

SPECIFIC CARDIOVASCULAR PROBLEMS IN AVIATION
- Conditions Which Influence G Tolerance
- Diseases Which Influence Tolerance of Hypoxia

SPECIAL MEDICAL PROBLEMS
- Hypertension
- Syncope and Lightheadedness
- Coronary Artery Disease
- Cardiac Arrhythmias
- Cardiac Trauma
- Cardiac Surgery

TRANSPORTATION OF THE CARDIAC PATIENT BY AIR

REFERENCES

THE CARDIOVASCULAR system has assumed progressively greater importance in the realm of aviation as man, in extending his accomplishments aloft, has exposed the flyer to increasing altitude and accelerative stress. It is in this context that the cardiovascular system often imposes the limiting factor upon the performance of aviators and, therefore, upon the performance of manned aircraft. For these reasons, a full understanding of physiological and pathological cardiovascular factors in aviation is essential to the Flight Surgeons charged with the selection and with the physical well-being of aviators.

CARDIOVASCULAR PHYSIOLOGY

The heart is a relatively efficient muscular pump strategically placed within the bony thorax. As with all other muscles, mechanical contraction of the actinomyosin complex is stimulated by electrical depolarization of each part of the myocardial syncytium. The self-propagating sequence of electrical depolarization originates in the sinoatrial node, between 50 and 100 times each minute, and is transmitted via the atria and the atrioventricular node to the ventricles. Extremely rapid transmission via the bundle of His and the Purkinje network leads to nearly simultaneous contraction of the entire ventricular muscle. This electrical activity, when recorded at points relatively distant from the heart, constitutes the electrocardiogram. However, the electrical activity, although causally related to the mechanical contraction of the myocardium, gives little direct information about the power of the heart.

The mechanical events of the cardiac cycle are briefly summarized below. Ventricular systole, which begins in both ventricles nearly simultaneously, rapidly elevates ventricular pressures above atrial pressures and leads to closure of the atrioventricular valves. Thus, the first heart sound, which represents the slightly asynchronous closure of the mitral and tricuspid valves, marks the start of isometric contraction. The rapidly rising ventricular pressures shortly exceed the pressure in the ascending aorta and pulmonary artery, opening the respective semilunar valves. Blood is expelled during the remainder of ventricular systole at normal peak systolic pressures of 120 and 20mm Hg in the aorta and pulmonary artery respectively. The ventricles normally expel about 50 percent of their contents prior to the start of relaxation. The events of ventricular diastole then occur. Ventricular pressures fall below vascular pressures; the semilunar valves perforce close, producing the aortic and, approximately 0.01–0.05 seconds later, the pulmonic second sounds; isometric relaxation ensues until ventricular pressure falls below the pressure in the slowly filling atria; and the atrioventricular valves open. The blood which has accumulated in the right and left atria, from the systemic and pulmonic veins respectively, passively flows into the ventricles. This flow reduces atrial pressure until electrical activation of the atria leads to mechanical atrial systole. A second rise in atrial pressure and a second period of rapid ventricular filling result. It is these two phases of rapid ventricular filling which give rise to the third and fourth heart sounds heard in many vigorous normal subjects and which, when accentuated, are designated as gallop cadences in congestive heart failure.

The pulmonary vessels constitute a low tension-low resistance bed with an extensive capillary network encompassing the pulmonary alveoli. An efficient transfer of gases between the alveoli and hemoglobin occurs prior to the coalescence of the vascular channels into the four primary pulmonary veins. These conduct blood with a Po_2 of 100mm Hg and a Pco_2 of about 40mm Hg into the left atrium. It is the low blood pressure as well as the distensibility of the lungs which leads to striking phasic alterations in pulmonary hemodynamics during normal or exaggerated respiratory movements. Clinically, perhaps the two most apparent manifestations of this are the accentuated splitting of the aortic and pulmonic second sounds during normal inspiration and the slight normal inspiratory decrease in systolic blood pressure (called a paradoxical pulse when accentuated as in constrictive pericarditis).

The pulmonary vascular resistance is affected by a variety of stimuli, including the autonomic nervous system and serotonin. Two factors which assume importance in relation to aviation are the pulmonary vasoconstriction which accompanies low inspired oxygen tensions and the reduced perfusion of the upper portions of the lung which exists normally and which is accentuated during positive acceleration.

The systemic circulatory system includes several segments. The large elastic arteries serve to assist flow during diastole by elastic recoil. The muscular arteries are conduits with a relatively limited capability of constriction and dilation. The arterioles, which produce a further increase in the size of the vascular bed and a concomitant reduction in the perfusion pressure, are the major site of peripheral vascular resistance, largely through the influence of autonomic or humoral vasoconstrictor or vasodilator stimuli. The capillaries, small and vast in number, are perfused at low pressures. In many organs they have the capacity of opening or closing some pathways to satisfy the varying metabolic needs of the tissues. Tissue hypoxia and the resulting products of anaerobic metabolism are potent stimuli to local dilation and increased perfusion through the capillary beds.

From the capillary bed, venules merge to form successively larger veins. Flow thence occurs to the right atrium via the venae cavae. The venous bed has definite but limited powers of active constriction which are important in man's ability or inability to adapt to a normal erect posture, and which, with arteriolar tone, play an important but unquantified role in the adaptation to abnormally high G stress. However, the thin-walled and distensible veins are the site of extensive pooling of blood during normal standing, a process which is exaggerated during acceleration.

Myocardial metabolism is almost exclusively aerobic, although there is some evidence that during hypoxia anaerobic glycolysis may supply some of the energy which is stored in adenosine triphosphate. This dependence upon aerobic metabolism precludes oxygen debts of the type which occur in skeletal muscle. For this reason the heart is quite susceptible to tissue hypoxia. The myocardium normally metabolizes carbohydrates or nonesterified fatty acids, but it can utilize lactate, ketones, and amino acids depending upon the concentration of the substrates in coronary arterial blood. It is in part this diversity which makes hypoglycemia less critical in terms of myocardial function than it is for cerebral function.

Coronary arterial blood flow depends upon the caliber of the vessels, the pressure in the aorta at the coronary orifices, and the compression of the vessels by the contracting myocardium. Among the potent stimuli for coronary vasodilation are epinephrine, sympathetic nervous system stimulation, and hypoxia. All of these factors tend to protect the heart against the effects of hypoxia on the oxidative myocardial metabolism. Coronary perfusion is reduced by compression by ventricular muscle during systole, and occurs more efficiently during the longer diastole. This dependence upon the aortic blood pressure and the central origin of the coronary vessels provide substantial protection to the heart against the altered hemodynamics of abnormal accelerations. As a result, the heart is infrequently the source of failure during the exposure of men to the stresses of aviation.

Cerebral blood flow and cerebral oxygen supply are more frequently critical during the stresses of military aviation than is coronary blood flow. Several protective mechanisms are important. Not only is the brain a passive recipient of increased blood because of increases in cardiac output and in systemic vascular resistance, but the cerebral vessels themselves are capable of marked changes in size and in resistance to flow. Arterial hypoxia and arterial hypercapnia are among the most potent stimuli for cerebral vasodilation. Conversely, cerebral blood flow is slightly reduced by hyperoxia and is markedly reduced by the hypocapnia which is produced by hyperventilation. Since hypoxia leads to hyperventilation, and since hyperventilation leads to hypocapnia, hypoxia exposes the cerebral vessels both to vasodilator and vasoconstrictor impulses. However, experimental work has shown that, in man, hypoxia leads to a protective increase in cerebral blood flow despite the associated hypocapnia.

The total output of the heart is approximately 3.5 L/min/M^2 of body surface area from each ventricle but may reach 20 L/min/M^2 during strenuous exertion. The left ventricular output is partitioned under normal circumstances approximately as follows: cerebral blood flow, 20 percent; coronary blood flow, 5 percent; renal blood flow, 25 percent; splanchnic and hepatic blood flow, 25 percent; and the remaining tissues, 25 percent. This partition is substantially altered with any stressful situation, largely because of autonomically induced vasoconstriction. Thus, fright, exertion, and other stresses divert part of the renal and splanchnic flow to the muscles, brain, and heart. Similar vasoregulatory mechanisms act during positive acceleration and during hypoxia to help maintain cerebral and coronary flow.

Regulation of cardiac output and blood pressure is dependent upon many interrelated physical, humoral, and nervous mechanisms. Basic to all is an adequate return of venous blood to the heart by the mechanisms of gravity, intrinsic venous tone, normal venous valves, massage of the veins by skeletal muscles, and a sufficient pressure in the capillary bed.

Recent studies have shown that the normal heart responds to most stresses by sympathetic nervous system stimulation of heart rate and of myocardial contractility. The result is an increase in ventricular stroke volume, at the expense of the residual volume, combined with a variable increase in the heart rate. These studies have not demonstrated the concordant increase in ventricular diastolic pressure or volume which would be required if the classical Starling's law is the only factor operative in the augmented flow. However, a passive shift from the erect to the supine position has been shown to alter ventricular systolic output as first the right and then the left ventricular filling pressure increases. In addition, the associated increase in right atrial filling produces cardiac acceleration by means of the Bainbridge reflex. Thus, it may be postulated that the adjustment of cardiac output during changing G states will be the resultant both of altered filling pressures and of epinephrine-norepinephrine stimulation. Whereas Starling's law is certainly a valid generalization, it should be stressed that markedly different ventricular function curves may exist for a given normal heart depending upon autonomic nervous system activity, nutrition, and other factors. Furthermore, many cardiovascular diseases profoundly alter the cardiac regulatory mechanisms, producing still different ventricular function curves.

The presence of arterial hypoxia also influences cardiac regulation. Hyperventilation induced by hypoxia produces sinus tachycardia by vagal inhibition. Sympathetic nervous stimulation increases the tachycardia, increases stroke volume, and also tends to produce selective vasoconstriction. The resulting increases in cardiac output and in arteriolar tone serve to counteract the concomitant locally mediated peripheral vasodilation.

The arterial pressure depends essentially upon the relationship between total flow and the peripheral arteriolar resistance to flow and, therefore, upon many of the mechanisms discussed above. Other structures which play a major role in blood pressure regulation are receptors in the aorta and in the carotid sinus. Both subserve dual functions as baroreceptors and chemoreceptors with influences upon the relation between arteriolar resistance and cardiac output, and upon the control of respiration. The former is manifested by reflex arcs which produce tachycardia, increased stroke volume, and renal and splanchnic vasoconstriction when arterial pressure falls. Conversely, a rise in arterial pressure tends to produce slowing of the sinoatrial pacemaker, decreased cardiac output, and peripheral vasodilation. It is here that excess vagal activity, leading to bradycardia, sinus arrest, and other arrythmias, originates during exposure to "red out" or negative acceleration conditions.

Local tissue requirements also mediate, probably chemically, local arteriolar, capillary, and venous resistance, while the net peripheral resistance in the entire vascular bed is maintained within physiological limits by centrally controlled mechanisms. The response of blood pressure to generalized and severe hypoxia depends upon the balance between the locally mediated arteriolar dilator stimuli and the vasoconstrictor impulses from the baroreceptors and the sympathetic nervous system. As tissue hypoxia persists, or becomes more severe, peripheral vascular collapse ensues. This usually follows cerebral dysfunction and precedes cardiac failure.

The physiological aspects described above have several direct applications to and implications for aerospace medicine.

Positive Acceleration

The primary cardiovascular effect of positive acceleration is an alteration in the normal hydrostatic relationships in the blood vessels. Positive acceleration, by adding to the "weight" of blood, leads simultaneously to increased pressure in the arterial and venous system in the lower half of the body, and to reduced blood pressure and filling of vessels in the upper half. Direct results are petechiae and, if sustained gravity forces are experienced, edema in areas of venous pooling. As the blood pressure falls, the carotid and aortic baroreceptors become active. These, stimulated by reduced intraluminal pressure, lead to cardiac acceleration, transiently increased stroke volume, and vasoconstriction in the splanchnic, cutaneous, renal, and probably other vascular beds. Cardiac output may increase but usually decreases slightly as the heart size decreases because of reduced venous return. These compensatory mechanisms become active only after several seconds, accounting for man's slightly better tolerance to gradually increasing acceleration than to abrupt change. Moreover, at peak loads of less than 5 G, transient narrowing of visual fields may be noted prior to complete cardiovascular compensation. These compensatory mechanisms permit a tolerance of about 5 G positive acceleration in normal subjects. At about this level venous pooling so restricts cardiac output that further compensatory measures are ineffective; the weight of the hydrostatic column between the aortic valve and the retinal and cerebral vessels cannot be overcome by the systolic pressure in the aorta; and peripheral retinal, central retinal, and cerebral blood flows cease sequentially. It should be noted that failure during acceleration represents a failure of peripheral circulation rather than of the vigorously beating, although almost empty, heart with its strategically situated coronary blood supply. An invariable feature of positive acceleration is sinus tachycardia, but instances have been reported of ventricular premature systoles, atrial premature systoles, and of atrial fibrillation. The specific effects of acceleration on the pulmonary circulation are of minor significance, but preferential flow through dependent portions of the lung often produce a mild ventilation–perfusion imbalance with a slight decrease in systemic arterial oxygen tension.

Antiblackout, or *G suits,* have been developed to further increase human tolerance by providing external compression of portions of the vascular bed. This compression, most frequently performed with tightly applied inflatable bladders, reduces venous pooling and makes peripheral and splanchnic arteriolar tone more effective. In this way cerebral and retinal perfusion are more efficiently maintained.

Negative Acceleration

With negative acceleration the same physical factors are operative, but quite different physiological effects occur. When the vector of inertial and gravitational force is oriented toward the head rather than toward the feet, the hydrostatic effect is to remove blood from the lower portions of the body and to pool blood under high pressures in the upper portions. Stimulation of baroreceptors leads to vagal discharge with cardiac slowing and even to shifting of the atrial pacemaker, sinus arrest with nodal escape, atrioventricular block, or other arrhythmias. The reflex vasodilation which also occurs is of limited benefit, but combined with a reduced cardiac output, does reduce aortic systolic blood pressure. For limited periods of negative acceleration no ill effects result to parts deprived of blood, but the intense intravascular pressure in cephalad areas may lead to edema, or to extravasations of blood. The high arteriolar and venous pressures lead to reduced flow, cerebral tissue hypoxia, unconsciousness, convulsions and, if prolonged, eventually to death at about minus 3 G. More intense acceleration in this direction may lead to more rapid cerebral anoxia, to intraocular hemorrhage, or even to cerebral hemorrhage. The latter is rare, largely because of the shielding calvaria and the increase in extravascular cerebrospinal fluid hydrostatic pressure. It is to be noted that man, accustomed as he is to positive 1 G, is poorly equipped to compensate for negative acceleration stress. Moreover, external compression by a G suit is of no benefit.

Transverse Acceleration

In contrast to acceleration along the longitudinal axis of the body, man has shown a remarkably high tolerance for gravity loads when he is oriented transversely to the axis of the gravity vector. This improved tolerance is largely related to the reduced length along the axis of acceleration of all hydrostatic columns.

Weightlessness

This physiologically unnatural state has recently been subjected to limited study in Projects Mercury and Gemini and will assume greater importance in the future of space-exploration. Gemini flights, especially, have shown that, as predicted, cardiovascular response (as measured by resting pulse rate and blood pressure, as well as tilt table studies) showed some orthostatic intolerance for 24 to 48 hours following a two-week mission in the weightless state. However, during the 14 weightless days of the flight, cardiovascular response to a given calibrated workload was relatively constant for a given individual, and no significant decrement in the physical condition of either pilot was noted. Further information about cardiovascular response to weightlessness will be obtained from studies conducted in the manned orbiting laboratories now scheduled for utilization within a short period of time.

Hypoxia

Man's responses to hypoxia have been considered in part. With decreasing alveolar and arterial Po_2 peripheral vasodilation occurs, but the effects of this are delayed and modified by the activity of the baroreceptors. The chemoreceptors simultaneously respond to the reduced arterial oxygen tension by reflexly inducing hyperventilation. This, in turn, may produce severe hypocapnia which is an additional important stimulus to systemic vasodilation. Concomitantly, tachycardia and augmented stroke volume lead to increased cardiac output and thereby tend to maintain the systemic blood pressure. However, at arterial oxygen tensions of about 50 to 60mm Hg, the blood pressure falls because of intense and generalized locally mediated vasodilation. Circulatory collapse thus may appear suddenly in unacclimated man at altitudes of about 20,000 feet. Here again, the peripheral circulation and the brain fail prior to failure of the heart itself. A direct corollary of this is that the effects of hypoxia and of positive acceleration may be additive. In addition, the dangers of reduced pulmonary or blood capacity to transfer oxygen and of undue exertion at altitude are obvious.

With acclimatization, man's ceiling is extended to about 25,000 feet, largely through changes in pulmonary ventilatory capacity and in the mass of hemoglobin within the body. Right ventricular hypertrophy may occur after prolonged exposure due, in part, to pulmonary vasoconstriction induced by hypoxemia. However, no studies have shown any such chronic compensatory changes in aviators.

One hundred percent oxygen extends man's capability to approximately 35,000 feet where, again, alveolar P_{O_2} reaches marginal levels. Positive pressure applied externally to the oxygen extends the feasible altitude only by about 8,000 feet. This limitation is imposed by the fact that a pressure differential between the lung and the environment exceeding about 25mm Hg, significantly impedes venous return to the heart. Thus, to permit flights above this altitude for a prolonged period, encompassing garments and pressurized cabins have been developed which will permit both a life-sustaining partial pressure of oxygen and tolerable pressure differentials.

Such suits and pressurized aircraft cabins yield the possibility of an abrupt loss of inspired P_{O_2} due to explosive decompression. Here, apart from the associated barotrauma, the duration of consciousness is dictated by the circulatory factors of rapid depletion of alveolar oxygen, the five- to eight-second period for circulation from lung to brain, and the almost nonexistent oxygen reserve in the brain. Death then ensues within five minutes from anoxia alone.

Reduced Atmospheric Pressure

The cardiovascular system is intrinsically relatively insensitive to altered ambient pressures. However, it is within the bloodstream that gaseous nitrogen accumulations produce potentially lethal effects. Pulmonary, cerebral, coronary, and other vascular beds are the site of disabling aeroembolism. Pulmonary congestion and edema may lead to dyspnea and to severe coughing. These factors, combined with others, may lead to acute heart failure. There are no specific cardiovascular measures to be taken other than preoxygenation as prophylaxis, and bodily recompression as treatment.

CLINICAL EVALUATION OF THE AVIATOR

The stresses discussed above have been considered as they affect the normal cardiovascular system. There are, however, many conditions which would be classified as minor variations from normal in nonaviation practice, as well as many disease states, which significantly detract from the ability of an individual to tolerate these stressful situations.

The detection and study of these factors is perhaps the single most challenging task of the Flight Surgeon. The diagnostic tools available are well known, but will be briefly considered in order of increasing complexity. As is often true, increased complexity of diagnostic methods implies decreased usefulness.

Medical History

An overwhelming facet of the diagnostic inquiry is the willingness of the subject to furnish facts. A strong motivation towards aviation may proportionately reduce the information obtainable unless the history is elicited with the utmost sympathy. In addition to the usual, often perfunctory, inquiries about dyspnea, chest pain, and other symptoms, special attention should be directed to syncope, symptoms of orthostatic intolerance or of carotid sinus sensitivity, palpitation of any nature, and discomfort or pain in any of the areas to which cardiac pain may radiate. Although definitive knowledge of the genetic relationships is lacking, several important diseases have familial trends which should be sought. Hypertension, coronary artery disease occurring in relatives under the age of 50, rheumatic heart disease, and congenital heart disease are among these.

The previous history of illness may be of extreme importance in instances of suspected congenital heart disease, rheumatic heart disease, and in the evaluation of orthostatic intolerance. The latter has been observed for months following certain viral illnesses and may be the important residual of a previous peripheral neuropathy.

Physical Examination

An examination of the heart alone does not suffice. Rather, the heart is merely part of the entire cardiovascular system. In the process of a general examination, careful note must be taken of all peripheral vessels, the jugular venous pulse and pressure, and of the retinal arterioles. The carotid arteries are particularly well situated to give information about the pulse contour as well as to assist in differentiating systole and diastole during cardiac auscultation.

The blood pressure should be obtained while sitting, while supine, and then immediately upon standing. The pressure tends to be about 10mm Hg higher in the left arm than in the right arm, and is probably a better reflection of central aortic pressure. The systolic and diastolic readings are determined respectively at the onset and at the disappearance of the Korotkoff sounds over the brachial artery. The blood pressure in the legs, determined by thigh compression and auscultation over the popliteal artery, is normally approximately 20mm Hg higher than that in the left arm. The lack of this difference is important in the detection of coarctation of the aorta.

Examination of the heart includes careful evaluation of heart size by palpation and percussion. A hyperdynamic apical impulse is seen frequently in the apprehensive normal subject, but when exaggerated strongly suggests left ventricular enlargement. A vigorous left parasternal thrust or bony protrusion may reveal right ventricular enlargement. The heart sounds, when examined by careful auscultation, may indicate any of the following: the abnormally and constantly wide temporal split between the aortic and pulmonary second sounds of atrial septal defect; the accentuated or decreased mitral first sound of mitral stenosis or incompetence respectively; the accentuated pulmonary valvular sound of pulmonary hypertension; the decreased aortic sound of aortic valvular disease; or the accentuated third and fourth sounds of increased left or right ventricular filling heard with patent ductus arteriosus and atrial septal defect respectively.

Auscultation for murmurs should include, in contradistinction to the usual practice, at least eight basic areas: (1) cardiac apex, (2) low left sternal margin, (3) midleft sternal margin, (4) second left, and (5) second right interspace parasternally, (6) left infraclavicular area, (7) carotid vessels, and (8) the area mesial to the left scapular margin. After preliminary identification of the heart sounds and of systole and diastole, auscultation need not be prolonged to be thorough.

Evaluation of a cardiac murmur is a frequent problem. It is to be noted at the outset that a diastolic murmur is invariably of pathological origin, and should be considered disqualifying for aviation in almost all instances. Systolic murmurs, on the other hand, occur in about half of normal healthy men as well as in a wide variety of disease states. It is this latter differentiation which taxes the capacity of the Flight Surgeon.

Systolic murmurs are of two basic types. Since ventricular pressure exceeds atrial, and left ventricular pressure exceeds right ventricular pressure, from the instant of the first heart sound until slighly after the second heart sound, defects between any of these chamber pairs will produce a murmur throughout systole: the pansystolic murmur of mitral incompetence, tricuspid incompetence, or ventricular septal defect. The presence of the period of isometric contraction has direct clinical application in the brief silent interval between the first heart sound and the onset of systolic murmurs originating from flow through the aortic or pulmonary valves or through a stenotic great vessel. Moreover, aortic and pulmonary flow increase and then decrease during a given ejection phase, producing a crescendo-decrescendo pattern in such murmurs. These are called systolic ejection murmurs, and are the type of murmur heard in aortic stenosis, pulmonary stenosis, and coarctation of the aorta. In addition, this type of murmur may originate at a normal pulmonary valve in many conditions characterized by rapid blood flow such as thyrotoxicosis, severe anemia, pregnancy, extreme exertion, and atrial septal defect of the secundum type. The differentiation of "innocent" from significant systolic murmurs depends primarily upon the absence of other cardiovascular abnormalities. However, most "innocent" murmurs have the following characteristics: (1) an injection quality, (2) grade 3 or less in intensity (on a scale of 1–6), (3) pulmonary valvular origin, and (4) a tendency to vary in intensity with position, respiration, rest, and exercise. A minority of "innocent" murmurs are heard near the cardiac apex, but even here the above description is valid. Again, it should be stressed that many pathological states may have murmurs with all of the characteristics described; the final decision is based upon the cardiac company kept by the murmur.

Radiology

Complete cardiac evaluation cannot be performed without X-ray evidence of the size and contour of all the cardiac chambers and great vessels. This can only be accomplished by careful study of the postero-anterior, lateral, and oblique projections of the chest.

Electrocardiography

An electrocardiogram is required for all flight candidates and naval aviators on active duty. Aviators above the age of 30 should have annual electrocardiograms, and such are mandatory on their 25th, 30th, and 35th birthdays and yearly thereafter except when facilities are unavailable.

The electrocardiogram serves several functions. First, it may be the sole means of detecting such lesions as bundle branch block and Wolff-Parkinson-

White syndrome, and of determining the nature of an arrhythmia. Secondly, the electrocardiogram may provide confirmatory evidence of cardiac chamber enlargement. Furthermore, the electrocardiogram serves as an important screening test for asymptomatic significant coronary artery disease. Finally, the electrocardiograms now being obtained on all aviators will serve as important controls with which subsequently developing abnormalities may be compared.

Other abnormalities which are detected only by the electrocardiogram are briefly considered here. Minor T wave abnormalities, secondary R waves in Leads V_1 and V_2 associated with an abnormal duration of the ventricular complexes, prolongation of the P–R interval, and several basically slow variants of normal sinus rhythm may raise questions about the ability of a man to control an aircraft. Since each of these electrocardiographic findings may be a variant of the normal, a thorough evaluation of the individual is necessary before a decision is made. However, minor physiological T wave variations usually revert to normal several hours after a meal, following exercise, or following the oral administration of potassium. Small secondary R waves in the right precordial leads may be considered to be a normal variant if, after a thorough clinical examination, all else is normal. The high incidence of similar electrocardiographic changes and the frequent absence of loud murmurs in atrial septal defect make it especially important to rule out this lesion. A prolonged P–R interval, in the absence of rheumatic fever or other febrile illnesses, may physiologically revert to normal with a reduction in vagal tone produced by standing, the Valsalva maneuver, exercise, or atropine. These same measures will also abolish many of the physiological variants of a slow sinus rhythm.

More troublesome are asymptomatic persons with bundle branch block. Experience has shown that left bundle branch block often presages more serious manifestations of disease, whereas right bundle branch block is less likely to do so. Accordingly, if all else is normal, the person with right bundle branch block may be perfectly capable of withstanding the stresses of aviation. However, in most instances of left bundle branch block, it must be presumed that disease exists.

Other Diagnostic Procedures

The Flight Surgeon may resort to ballistocardiography, phonocardiography, and cardiac catheterization if the clinical need arises. However, the decision to ground an aviator will seldom be made solely upon evidence from these tests. Certain chemical and immunological analyses of blood composition usually are helpful, though ancillary, aids to diagnosis. The many well-recognized diagnostic aids in hypertension will be performed for therapeutic reasons, but usually will not influence the decision to ground an aviator.

SPECIFIC CARDIOVASCULAR PROBLEMS IN AVIATION

The natural medical history of the naval aviator begins at the time of his selection for flight training and ends with retirement or transfer to nonflying duties. Chronologically, his career can be roughly divided into four stages: (1) selection, (2) flight training, (3) operational and/or combat flying, and (4) military commander with restricted flight status. The cardiovascular problems which confront the Flight Surgeon are somewhat related to these different periods in the aviator's career.

Selection.—The initial selection of the candidate for flight training is based on rigid physical standards which have been meticulously defined. One aspect of the problem here is concerned with reliability of the tests used and their validation. More immediate problems have to do with the physical examination such as the evaluation of systolic murmurs, the problem of "borderline" hypertension, and the significance of minor cardiac arrhythmias. Using the broad meaning of the term, "selection" does not end with the candidate's admission to flight training but persists in the form of annual physical examinations and examinations following hospitalization. On these occasions the flyer is either reselected to continue flying or rejected because of some disability. A good general policy to follow is to make few recommendations for waiving a requirement at the initial selection but be guided thereafter by the concept of the "good of the service."

Training Period.—During flight training, and indeed during operational flying, frank cardiovascular disease develops infrequently but problems arise concerning such things as (1) the evaluation of chest pain, (2) functional disorders of the heart, chiefly arrhythmias, and (3) dizziness or ease of blackout.

Operational Period.—The aviator is subjected to the greatest stresses during the operational period of his career, but these stresses are also severe during certain stages of flight training. If these stresses induce circulatory failure, it is the peripheral circulation which is primarily involved in nearly every instance. This emphasizes the importance of adequate examination and testing. Unfortunately, this presents great difficulties because of the lack of reliable tests and because of the great individual variation in response from time to time.

Command Period.—At the age of 45, the aviator automatically is reassigned from service group I to service group III flight status. Thereafter, he is no longer subject to the stresses of operational flying. However, the effects of aging soon begin to appear and two of the most important medical problems are essential hypertension and coronary heart disease.

Conditions Which Influence G Tolerance

A wide variety of physiological factors have been shown to reduce man's tolerance to acceleration. Fatigue, extreme apprehension, minor degrees of hypoglycemia and dehydration are among these. Conditions such as high ambient temperatures, fever, and sunburn, which lead to peripheral vasodilation, also may adversely affect tolerance. Peripheral neuropathy, certain viral illnesses and conditions, such as mitral stenosis, which restrict cardiac output also have an undesirable effect on the normal mechanisms which compensate for acceleration stresses. In addition, venous thromboses, varicose leg veins and other lesions which interfere with normal venous return are potentially detrimental.

Paradoxically, significant hypertension should be mentioned in a discussion of reduced G tolerance. Although, theoretically, the elevated pressure should help to maintain retinal and cerebral blood flow, this may not be true, particularly with severe hypertension. First, the concomitantly elevated cerebral and retinal vascular resistance probably mitigates this advantage. In addition, recent studies have shown that hypertensives as well as orthostatic hypotensives do not show the normal rise in plasma pressor amine levels with a change from the supine to the erect position on a tilt table. Furthermore, it may be assumed that the limited normal ability to withstand negative acceleration is further reduced by a disease which is associated with high incidence of cerebrovascular accidents.

Even in the absence of extraneous influences, there is a wide individual variation in tolerance to acceleration. In extreme cases peripheral circulatory collapse, hypotension, and syncope in the erect position may occur even under the stress of 1 G. These variations among apparently normal persons probably reflect differences in sensitivity of the normal autonomic reflex area. Obviously, such overly sensitive individuals must be detected and eliminated from aviation programs. The separate disease entity of chronic orthostatic hypertension, which is characterized by anhydrosis, impotence, and hypotension, may rarely be encountered. The implications for aviation are the same.

Diseases Which Influence Tolerance of Hypoxia

An understanding of the intrinsic cardiovascular limitations to tolerance of hypoxia follows directly from the physiological considerations discussed previously.

Coronary artery disease, even in an asymptomatic stage, leads to direct decreases in myocardial perfusion under resting conditions. Moreover, the reduced coronary pliability decreases the effectiveness of coronary vasodilator stimuli during exercise or hypoxia. Concomitantly, the tolerance of the myocardium for hypoxemia is reduced. Under these conditions, the potential of coronary artery disease to produce serious arrhythmias, myocardial infarction, or decreased myocardial contractility is enhanced.

The fact that the cerebral oxygen supply is frequently the limiting factor in normal tolerance for hypoxia emphasizes the potential danger to the aviator posed by cerebrovascular stenosis. Both the direct stimulus to cerebral vasodilation produced by anoxia, and the response to systemic circulatory changes may be ineffective in augmenting cerebral blood flow.

Apart from the secondary alterations which hyperventilation due to hypoxia imposes upon cerebral and systemic blood flow, it is important to consider the effects of hyperventilation which may occur with severe apprehension or anxiety. The major change is a reduction in alveolar and arterial carbon dioxide tension. This, as discussed previously, is a powerful stimulus to cerebral vasoconstriction and, therefore, to a reduced cerebral oxygen supply. The decreased capillary and tissue oxygen tensions secondarily lessen the vasoconstriction, but the net effect is a reduction in cerebral blood flow approximately in proportion to the degree of hypocapnia. It is for this reason that hyperventilation is detrimental to man's ability to withstand either hypoxic or accelerative stress. Moreover, hyperventilation due to anxiety alone may, if severe and prolonged, lead to unconsciousness, probably due, in part, to reduced cerebral blood flow and cerebral oxygen supply.

Uncomplicated hypertension probably has little effect on adaptation to hypoxia, except for its statistical association with atherosclerosis of the coronary, carotid, and basilar arteries.

Congenital heart lesions with left to right shunting represent situations of decreased myocardial efficiency for a given level of systemic flow, whereas right to left shunts are associated with unphysiological arterial hypoxemia even under basal conditions. Either type of lesion, as well as pulmonary valvular stenosis, will be aggravated by increased pulmonary vascular resistance under hypoxic conditions.

Any obstructive or restrictive lesion in the airway from the nose through the alveoli must be considered to reduce tolerance for hypoxia as well as for barotrauma. Anemia or a reduced blood volume from any cause is incompatible with normal adjustment to reduced alveolar P_{O_2}, as are fever, high ambient temperatures, and sunburn.

All other forms of heart disease are potentially dangerous in the aviator. For example, paroxysmal arrhythmias, which a portion of individuals with the Wolff-Parkinson-White syndrome develop, may seriously affect the aviator's abilities if they occur under accelerative or hypoxic stress; rheumatic valvular lesions restrict normal cardiac regulation, or may, as in aortic stenosis, lead to sudden syncope, or may, as in aortic incompetence, compromise coronary perfusion; chronic or, temporarily, acute pericarditis may restrict cardiac output during any stress.

Finally, note should be made of environmental carbon monoxide. The steps which are taken to eliminate contamination of the aviator's atmosphere by carbon monoxide are usually effective, but occasional instances of toxicity are seen. However, it is inescapable that the 5–10 percent carboxyhemoglobin which is chronically present in the blood of a heavy cigarette smoker limits that man's tolerance of hypoxia.

SPECIFIC MEDICAL PROBLEMS

The processes of selection to which each prospective aviator is exposed imply that relatively few instances of rheumatic or congenital heart disease will be encountered in the established aviator. However, several cardiovascular problems will be seen frequently, particularly as individual aviators age.

Hypertension

The rigorous standards for selection as aviators exclude nearly all instances of significant hypertension in young aviators. Those young men with intermittently elevated diastolic pressures who manage, by sufficient relaxation, to pass the examination may account for the occasional cases encountered. It is to be noted that increasing evidence is available that men with intermittent diastolic hypertension during their earlier years have an unusually high incidence of hypertension later in life. Such men, therefore, may well represent a suboptimal population from which to select aviators. At present, however, the implication is clear that the established standards for selection should be carefully observed.

With increasing age, both the incidence and severity of hypertension increase, whether of "essential," "renal," or other origin. There is little statistical doubt that hypertension shortens life, but there is no sound reason to curtail the activities of an aviator beneath the age of 40 because of mild hypertension. In all likelihood, his operational flying career will be unaffected. However, diastolic pressures persistently above 110mm Hg are more likely to be associated with cerebral, retinal, and renal changes. Such patients warrant treatment with hypotensive drugs to preserve effective health. The associated effects of these drugs on normal cardiovascular regulatory mechanisms will usually mean that the start of treatment signals the end of operational flying duties.

Syncope and Lightheadedness

The occurrence of syncope represents a major hazard to the flyer and to his companions. For this reason, serious thought must be given to restricting the flying status of such men unless a clearly nonrecurring situation was responsible. Syncope due to hypoglycemia, carotid sinus hypersensitivity, hyperventilation, and orthostatic intolerance can be investigated and often reproduced by simple techniques. Vasovagal syncope, which is characterized by sinus bradycardia and peripheral vascular collapse, often occurs in an isolated instance, although some individuals have recurrent episodes. Precipitating factors such as hunger, fatigue, prolonged standing and psychic trauma may all be contributory. This type of syncope may be impossible to reproduce in the clinic and may, therefore, be a difficult problem for the Flight Surgeon. The final clinical and military decisions can only be reached after consideration of all aspects.

Closely related to the above are the various neurological causes of loss of consciousness. Complete evaluation of each man with syncope demands careful study by the Flight Surgeon from the point of view of each of several medical disciplines.

Coronary Artery Disease

First, it should be recorded that this is not a disease limited to men above the age of operational flying. Several studies have shown coronary atherosclerosis in up to 70 percent of military men killed before the age of 40.

In most instances there should be little question of the inadvisability of a man continuing as an aviator when he has angina pectoris or myocardial infarction. With careful evaluation of the chest pain, and of the associated symptoms, the diagnosis of symptomatic coronary disease will seldom be difficult. If the historical evidence is inconclusive in a man with possible angina pectoris, exercise with electrocardiographic followup will usually permit a diagnosis. Similarly, the associated changes on physical examination, the electrocardiogram, fever, leucocytosis and serum enzyme changes will seldom lead to error in the diagnosis of myocardial infarction. Occasional instances of acute pericarditis may closely mimic coronary disease, but the slightly different character of the pain, the history of recent respiratory illness, the electrocardiogram, and the appearance of a pericardial friction rub within the first 24 hours will usually permit correct diagnosis.

In contradistinction to the above is the large number of men with coronary disease who are asymptomatic during routine duty. These men may, however, represent a hazard during a stressful flight. Detection of these apparently healthy persons is notoriously difficult. There are undoubtedly many who escape detection, but this fact does not reduce the responsibility of the Flight Surgeon to decide upon the future in aviation of a man whose disease is detected.

Practically, the sole instrument of this detection process is the routine electrocardiogram. Rare instances of previous, apparently symptomless, myocardial infarction will be found. More frequently, abnormal T waves, frequent premature systoles, other arrhythmias, intraventricular block, or bundle branch block are the initial findings. As a general rule, all electrocardiographic abnormalities or significant changes in serial electrocardiograms should be considered invitations to a thorough examination of the individual. Some of these men will furnish historical information compatible with angina pectoris when confronted with electrocardiographic evidence. The majority, however, deny all symptoms. Experience at the Naval Aerospace Medical

Institute has shown that the single most reliable test in this situation is the electrocardiogram following strenuous exercise. The Harvard step test is performed with one up and down cycle on the 20-inch step every three seconds for two, three, four, and five minutes in turn. As with Master's step test, chest pain is cause for immediate cessation of exercise, and is direct evidence of a positive test even in the absence of electrocardiographic changes. The most widely used criterion of a positive test is the occurrence after exercise of increasing S–T segment deviation with increasing workload, compared with control tracings taken immediately prior to exercise. Other findings which may indicate myocardial ischemia are an increased frequency of premature beats, paroxysmal arrhythmias, and inversion of previously upright T waves. With the present lack of more definitive tests, it seems wise to abolish or, above the age of 40, to restrict the flying status of an aviator whose test is declared unequivocally positive by a physician experienced in reading electrocardiograms. Other studies, such as ballistocardiography, are of interest but have no value in establishing or ruling out the diagnosis. Serum levels of cholesterol and lipoproteins have been proved statistically valuable as means of determining a predisposition to coronary disease, but elevated levels do not suffice to make a diagnosis of coronary atherosclerosis.

As in hypertension, treatment of this disease does not offer hope of preserving flight status. Coronary vasodilators are systemic vasodilators and are, therefore, hazardous to the flyer. Digitalis is of no value in the man with asymptomatic coronary disease. Despite extensive study, there is no direct evidence in man that restriction or substitution of lipids in the diet prevents or reverses coronary atherosclerosis to a degree which would permit resumption of an aviator's career. However, reports of favorable changes in electrocardiograms and in prognosis suggest that therapeutic alteration of dietary lipids is beneficial to the patient with coronary disease.

Cardiac Arrhythmias

The problem of a wide variety of cardiac arrhythmias frequently arises in the evaluation of an aviator.

Normal cardiac rhythm may be arbitrarily defined as basically regular discharges of the sinoatrial pacemaker which activate the myocardium between 50 and 100 times each minute. Vagal stimulation leads to sinus bradycardia with rates less than 50 beats per minute. These slow rates are frequently encountered in physically robust young men. The high levels of vagal tone in such men may also produce the variation in heart rate during the respiratory cycle which is known as sinus arrhythmia. If vagal tone leads to marked slowing, other areas of the myocardium may temporarily assume the pacemaker function. Thus, a wide variety of basically slow physiological arrhythmias may be encountered. Almost without exception, shifting atrial pacemaker, atrial escape beats, nodal escape beats, and even ventricular escape beats have no pathological significance under these circumstances. However, the extremes of normal vagal tone may predispose to extreme cardiac slowing during negative acceleration.

Acceleration of the sinus pacemaker above 100 beats/min, called sinus tachycardia, usually reflects disease when it persists during basal conditions. Most frequently, minor viral illnesses will be discovered, but sinus tachycardia may be an early indication of more serious disease.

Paroxysmal arrhythmias occurring in the aviator represent a major potential danger to his ability to adapt to flying stresses. Precise identification and documentation of the rhythm is mandatory. If an electrocardiogram is not available, clinical identification may be possible from the rate, the type of regularity or irregularity, from the jugular venous pulse, and from the response to firm but not occlusive carotid sinus massage. Such arrhythmias, regardless of origin, should be considered disqualifying for aviation if symptoms of severe cardiovascular restriction occur during the attack. Other instances, if not associated with diagnosed coronary atherosclerosis or with other diseases, will have to be considered individually with a full knowledge of the clinical and aviation status of the patient.

A related problem is presented by the man with transient palpitations. Most frequently these are ventricular premature systoles, but electrocardiographic proof may be hard to obtain. These may occur in normal men during anxiety, or after excesses of alcohol, tobacco, or caffeine. Such persons can often be relieved of the symptoms by omission of the offending agents. Less frequently, premature systoles will be encountered which occur during, or are aggravated by, exercise. Men of this latter group seem, on physiological grounds, more likely to have myocardial or coronary disease. At the present time, however, there is no sound evidence which indicates that they should be grounded, unless the frequency of premature beats is so great that many brief episodes of paroxysmal tachycardia occur.

Cardiac Trauma

Cardiac trauma, which is unavoidably associated with many aspects of life, is particularly common in aviation. Penetrating wounds of the heart, if not quickly fatal, may become manifest either by profuse hemorrhage into a pleural cavity or by hemorrhage into the pericardium. The former should be suspected with any thoracic wound which is associated with hemothorax; the latter may be detected by the classical coexistence of shock and cardiac tamponade. Instances of extensive hemorrhage will usually require immediate surgery to suture the cardiac wound, but life can usually be sustained until definitive therapy is available by transfusion and aspiration of pleural collections of blood. Cardiac tamponade may be dramatically relieved by pericardiocentesis performed through the left costoxiphoid area. The precardial pressure–volume curve is sufficiently steep that the removal of even small amounts of fluid usually significantly improves venous return to the heart. If tamponade recurs, a second tap may be necessary prior to the availability of surgery. However, many such cases do not have recurrences and require no surgical intervention.

Myocardial ecchymoses and contusions should be suspected with all closed chest injuries. Symptoms, clinical signs and electrocardiographic changes similar to those of myocardial infarction may occur. The latter changes, if undetected during the acute illness, may lead to a later unwarranted diagnosis of coronary artery disease. Cardiac tamponade and arrhythmias are the most threatening manifestations of blunt cardiac trauma. However, rupture of the myocardium, the ventricular septum, or of valvular leaflets may occur acutely or several days later, and may require prompt surgical intervention.

Cardiac Surgery

The increasing frequency of precise surgical correction of congenital cardiovascular defects will have an impact on military aviation. Whereas many of these individuals must be considered to be normal for most activities, there is little direct evidence which supports or refutes their ability to withstand the stresses of aviation. Thus, at present, all such cases will have to be individually evaluated, probably during simulated flying conditions, before a decision is reached.

TRANSPORTATION OF THE CARDIAC PATIENT BY AIR

The story of the transport of sick and wounded by air during World War II, the Korean War, and Vietnam is one of the most remarkable in all military medical history. Nearly one-half million patients were carried by the Army Air Force in World War II, many desperately sick and wounded, yet it is estimated that fewer than 50 died as the result of flying stress. This, despite the fact that ofttimes there was little or no opportunity for the selection of cases. The present trend is toward air evacuation of the sick and wounded and the location of medical installations based on transport by helicopter and airplane. Except in the case of small aircraft in the combat zone, the transports are equipped and staffed to handle cardiovascular emergencies of all kinds.

However, there will still remain instances in which the Flight Surgeon will be expected to make the decision whether a patient with heart disease is fit to fly in an ordinary transport plane and without the benefit of medical care en route. The decision should be made on the basis of (1) evaluation of the cardiovascular status of the patient, (2) estimation of the flight stress to be encountered, and (3) consideration of the alternate possibilities, i.e., either postponing the trip or utilizing another means of travel. This subject is covered in a later chapter.

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CHAPTER 11

Gas Physiology in Naval Aerospace Medicine

By CDR C. L. Ewing, MC, USN, and LCDR M. Passaglia, Jr., MSC, USN

- Introduction—The Atmosphere
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  - Pulmonary Ventilation
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  - Operational Problems With Hypoxia
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  - Etiology
  - Predisposing Factors
  - Clinical Features
  - Differential Diagnosis

Clinical Management
Recompression Therapy
Prevention
Rapid and Explosive Decompression

INTRODUCTION—THE ATMOSPHERE

Table 11–1 presents the temperatures and pressures of the standard atmosphere in 1,000-foot increments to 100,000 feet, with altitudes presented in both feet and meters.

Table 11–2 presents the gaseous composition of the atmosphere at sea level. However, the relative or fractional concentrations remain constant from the surface of the earth to the outer limits of the atmosphere.

TABLE. 11–1. *Altitude-Pressure-Temperature Relationships Based on the U.S. Standard Atmosphere—1962*

Altitude		Pressure		Temperature	
Feet x 10^3	Meters	mm Hg	psia	°F	°C
0	0	760.0	14.7	59.0	15.0
1	304.8	732.9	14.2	55.4	13.0
2	609.6	706.7	13.7	51.9	11.0
3	914.4	681.2	13.2	48.3	9.1
4	1,219.2	656.4	12.7	44.8	7.1
5	1,524.0	632.4	12.2	41.2	5.1
6	1,828.8	609.1	11.8	37.6	3.1
7	2,133.6	586.5	11.3	34.0	1.1
8	2,438.4	564.6	10.9	30.5	−.8
9	2,743.2	543.4	10.5	26.9	−2.8
10	3,048.0	522.8	10.1	23.4	−4.8
11	3,352.8	502.8	9.7	19.8	−6.8
12	3,657.6	483.5	9.3	16.2	−8.8
13	3,962.4	464.8	9.0	12.7	−10.7
14	4,267.2	446.6	8.6	9.1	−12.7
15	4,572.0	429.1	8.3	5.5	−14.7
16	4,876.8	412.1	7.9	2.0	−16.7
17	5,181.6	395.7	7.7	−1.6	−18.7
18	5,486.4	379.8	7.3	−5.0	−20.6
19	5,791.2	364.4	7.0	−8.7	−22.6
20	6,096.0	349.5	6.8	−12.3	−24.6
21	6,400.8	335.2	6.5	−15.8	−26.6
22	6,705.6	321.3	6.2	−19.4	−28.5

TABLE 11-1. *Altitude-Pressure-Temperature Relationships Based on the U.S. Standard Atmosphere—1962*—Continued

Altitude		Pressure		Temperature	
Feet x 10³	Meters	mm Hg	psia	°F	°C
23	7,010.4	307.9	5.9	−22.9	−30.5
24	7,315.2	294.9	5.7	−26.5	−32.5
25	7,620.0	282.4	5.5	−30.0	−34.5
26	7,924.8	270.3	5.2	−33.6	−36.5
27	8,229.6	258.7	5.0	−37.2	−38.4
28	8,533.4	247.4	4.8	−40.7	−40.4
29	8,839.2	236.6	4.6	−44.3	−42.4
30	9,144.0	226.1	4.4	−47.8	−44.4
31	9,448.8	216.1	4.2	−51.4	−46.3
32	9,753.6	206.4	3.9	−54.9	−48.3
33	10,058.4	197.0	3.8	−58.5	−50.3
34	10,363.2	187.9	3.6	−62.1	−52.3
35	10,668.0	179.3	3.5	−65.6	−54.2
36	10,972.8	170.9	3.3	−69.2	−56.2
37	11,277.6	162.9	3.2	−69.7	*−56.5
38	11,582.4	155.4	3.0	...	...
39	11,887.2	148.1	2.9	...	...
40	12,192.0	141.2	2.7	...	...
41	12,496.8	134.5	2.6	...	...
42	12,801.6	128.3	2.5	...	...
43	13,106.4	122.3	2.4	...	...
44	13,411.2	116.6	2.3	...	...
45	13,716.0	111.1	2.1	...	...
46	14,020.8	105.9	2.0	...	...
47	14,325.6	100.9	1.9	...	...
48	14,630.4	96.3	1.9	...	...
49	14,935.2	91.8	1.8	...	...
50	15,240.0	87.5	1.7	...	...
51	15,544.8	83.4	1.6	...	...
52	15,849.6	79.5	1.5	...	...
53	16,154.4	75.8	1.5	...	...
54	16,459.2	72.3	1.4	...	...
55	16,764.0	68.9	1.3	...	...
56	17,068.8	65.7	1.3	...	...
57	17,373.6	52.6	1.2	...	...
58	17,678.4	59.7	1.2	...	...
59	17,983.2	56.9	1.1	...	...
60	18,288.0	54.2	1.0	...	...
61	18,592.8	51.7	0.9	...	...
62	18,897.6	49.3	0.9	...	...
63	19,202.4	46.9	0.9	...	...
64	19,507.2	44.8	0.8	...	...
65	19,812.0	42.7	0.8	...	...
66	20,116.8	40.7	113.4	...	...

* See footnote at end of table.

TABLE 11-1. *Altitude-Pressure-Temperature Relationships Based on the U.S. Standard Atmosphere—1962*—Continued

Altitude		Pressure		Temperature	
Feet x 10^3	Meters	mm Hg	psia	°F	°C
67	70, 421.6	38.8	108.1	....	....
68	20, 726.4	37.0	103.1	....	....
69	21, 031.2	35.3	98.3	....	....
70	21, 336.0	33.7	93.7	....	....
71	21, 640.8	32.1	89.4	....	....
72	21, 945.6	30.6	85.3	....	....
73	22, 250.4	29.2	81.3	....	....
74	22, 555.2	27.9	77.6	....	....
75	22, 860.0	26.6	74.0	....	....
76	23, 164.8	25.4	70.6	....	....
77	23, 469.6	24.2	67.4	....	....
78	23, 774.4	23.1	64.3	....	....
79	24, 079.2	22.0	61.3	....	....
80	24, 384.0	21.0	58.5	....	....
81	24, 688.8	20.1	55.8	....	....
82	24, 993.6	19.1	53.3	....	....
83	25, 298.4	18.3	50.9	....	....
84	25, 603.2	17.4	48.6	....	....
85	25, 908.0	16.6	46.4	....	....
86	26, 212.8	15.9	44.3	....	....
87	26, 517.6	15.2	42.3	....	....
88	26, 822.4	14.5	40.3	....	....
89	27, 127.2	13.8	38.5	....	....
90	27, 432.0	13.2	36.8	....	....
91	27, 736.8	12.6	35.1	....	....
92	28, 041.6	12.0	35.6	....	....
93	28, 346.4	11.5	32.0	....	....
94	28, 651.2	11.0	30.6	....	....
95	28, 956.0	10.5	29.2	....	....
96	29, 260.8	10.0	27.9	....	....
97	29, 565.6	9.6	26.6	....	....
98	29, 870.4	9.2	25.5	....	....
99	30, 175.2	8.7	24.4	....	....
100	30, 480.0	8.4	23.3	....	....

*Temperature remains constant above this level up to about 22 miles.

Table 11-2. *Composition of the Dry Atmosphere*

Gas	*Volume percent*
Nitrogen	78.09
Oxygen	20.95
Argon	0.93
Carbon dioxide	0.03
Neon	1.80×10^{-3}

Table 11–2. *Composition of the Dry Atmosphere*—Continued

Gas	*Volume percent*
Krypton	1.00×10^{-4}
Helium	5.24×10^{-4}
Hydrogen	5.00×10^{-5}
Xenon	8.00×10^{-6}

The most common major divisions of the atmosphere are the inner and outer (exosphere) atmospheres. The former extends from the surface of the earth to an altitude of approximately 600 miles, and the latter extends from the 600-mile level to the outer limits of approximately 1,200 miles. The force of gravity and the radiation of the sun affect the molecular disposition within these layers; hence, the atmosphere becomes less dense with increasing altitudes.

The inner layer of the atmosphere is further subdivided into three zones:

1. The troposphere extends from sea level to the tropopause and is the area characterized by decreasing temperature with altitude, convection currents, and variable moisture content. The tropopause, within which temperature is constant, varies with latitude and seasons, averaging about 30,000 feet at the poles and about 56,000 feet over the equator. It acts as a water vapor trap.

2. The stratosphere extends from approximately 8 to 50 miles above the earth. Due to the sun's radiation, the greatest concentration of ozone is formed in this layer. The major importance of the ozone layer is that it absorbs most of the radiation of wavelengths shorter than 2,900 Angstrom Units.

3. The ionosphere begins at a height of about 50 miles and extends out to a distance of approximately 600 miles. The ionosphere can be further subdivided into four layers: D; E or Kennelly; Heaviside layer; F or Appleton layer, which is subdivided into the F_1 and F_2 layers.

A summary of the major properties of the atmosphere, revised in accordance with recent studies involving satellite drag and ion traps, is as follows:

There is a mixture of oxygen and nitrogen up to about 120 km, a layer consisting predominantly of atomic O_2 between that height and 1,000 km, a layer of helium from 1,000 to 2,500 km, and a hydrogen atmosphere extending out into interplanetary space above 2,500 km (Jastrow, 1962).

In addition to the gas layers themselves, there are other properties of the atmosphere in which the aviator is interested. A few of these considerations are listed here. Others are listed in the section on limiting altitudes (Strughold, 1960).

1. At about 25 km (16 miles), the air, due to its low density, can no longer be utilized for cabin pressurization; instead a sealed cabin is needed.
2. Forty km (24 miles) is the upper limit of the region of atmospheric absorption of cosmic rays, which are thereafter encountered in primary form.
3. Forty-five km (28 miles) is the upper limit of the region of atmospheric absorption of solar ultraviolet (which is absorbed within the altitude range of the ozonosphere) at 20 to 45 km.
4. The 50-km (30-mile) level is the upper limit for aerodynamic lift and navigation, even for the fastest winged craft.
5. At about 100 km (60 miles), the rarified air ceases to scatter light, leading to the so-called "darkness of space," and ceases to propagate sound, resulting in the "silence of space."
6. One hundred and twenty km (75 miles) is beyond the meteorite absorbing region of the atmosphere. It is, for all practical purposes, the upper limit for aerodynamic friction heat. Above here the hull temperature is determined exclusively by solar radiation.
7. At 200 km (120 miles) air resistance approaches zero but layers of atomic oxygen, then helium, and then hydrogen remain before reaching true interplanetary spatial conditions.

RESPIRATORY PHYSIOLOGY

In chapter 10, the cardiovascular aspects of respiratory physiology were discussed. Gas physiology is one of the cornerstones of aerospace medicine, and a great deal of work has been done in this field in connection with high-altitude military and civilian aircraft development as well as in support of manned space flight. The purpose of this chapter is not to present a complete compendium of this information but rather a skeleton upon which the interested Flight Surgeon will flesh in detail by his personal reading.

The major functions of respiration are to transport alveolar oxygen to the tissues and to transport tissue carbon dioxide back to the lungs. The process is effected by transporting gases from the exterior through the upper respiratory tract and trachea to the alveoli; letting the gases of the alveoli and pulmonary capillary blood reach equilibrium with each other; transporting the arterial blood to tissue where tissue gases reach equilibrium with arterial gases in the capillaries; and returning the blood to the lungs to repeat the process.

Individual cells within the tissues of the body are basically fluid in composition and as such are essentially incompressible. Pressure applied uniformly to a tissue surface thus is readily transmitted throughout the tissue and to adjoining structures. The important effects of changes in the pressure environment, therefore, arise not from cellular distortion but rather from the change in pressure of gases contained within the body. The manner in which changes in gas pressure affect the body can be expressed in terms of the following classical laws of gas mechanics.

Boyle's Law states that the volume of a gas is inversely proportional to its pressure, temperature remaining constant. This means that at 18,000 feet, where the pressure is approximately half that of sea level, a given volume of gas will attempt to expand to twice its initial volume in order to achieve equilibrium with the surrounding pressure.

Charles' Law states that the pressure of a gas is directly proportional to its absolute temperature, volume remaining constant. The contraction of gas due to temperature change at altitude in no manner compensates for the expansion due to the corresponding decrease in pressure.

Dalton's Law of Partial Pressures states that each gas in a mixture of gases behaves as if it alone occupied the total volume and exerts a pressure, its partial pressure, independently of the other gases present. The sum of the partial pressures of the individual gases is equal to the total pressure. Using this law, one can calculate the partial pressure of a gas in a mixture simply by knowing the percentage of concentration in that mixture.

Henry's Law states that the weight of a gas absorbed by a given liquid with which it does not combine chemically is directly proportional to the pressure of the gas.

Graham's Law. The relative rates of diffusion of gases under the same conditions of temperature and pressure are inversely proportional to the square roots of the densities of those gases. Gases with smaller molecular weights will diffuse more rapidly.

The four principal gases of interest in aerospace medicine are oxygen, nitrogen, carbon dioxide, and water vapor.

Pulmonary Ventilation

Ventilation is a cyclic process by which fresh air or gas mixture enters the lung and pulmonary air is expelled. The inspired volume is greater than the expired volume because the volume of O_2 absorbed by the blood is greater than the volume of CO_2 which is released from the blood. Since gas exchange occurs solely in the alveoli and not in the conducting airways, the estimation of alveolar ventilation rate (e.g., the amount of gas which enters the alveoli per minute) is the most important single variable of ventilation. Pulmonary ventilation is not perfectly distributed since normal lungs do not behave like perfect mixing chambers nor is the pulmonary capillary network evenly distributed throughout the lungs. In normal healthy individuals, there is strong evidence to suggest that uneven ventilation and uneven distribution of blood flow to different parts of the lungs do occur. Ventilation must be readjusted regionally to match the increased or decreased blood flow or some of the alveoli will be relatively under- or overventilated. The even distribution of pulmonary capillary blood flow is as important as an even distribution of inspired air or other gas mixture to the alveoli for normal oxygenation of the blood.

Gaseous Diffusion

Respiratory gas exchange in the lungs is accomplished entirely by the process of simple diffusion. The direction of movement of the molecules and the amount depend upon the difference in partial pressure on both sides of the alveolar membrane so that normally molecular oxygen moves from a region of higher partial pressure to one of lower partial pressure. The volume of gas which can pass across the alveolar membrane per unit time at a given pressure is the diffusing capacity of the lungs.

The diffusing capacity is not only dependent on the difference in partial pressure of the gas in the alveolar air and pulmonary capillary blood, but is also proportional to such factors as the effective surface area of the pulmonary vascular bed; inversely proportional to the average thickness of the alveolar membrane; and proportional to the solubility of the gas in the membrane. The normal values for the diffusing capacity range 20–30ml O_2/min/mm Hg for normal young adults.

Pulmonary Capillary Blood Flow

Inasmuch as the major function of the lungs is gas exchange, the pulmonary capillary blood flow must be adequate in volume and well distributed to all of the ventilated alveoli. Underperfusion of poorly ventilated alveoli can become a serious matter during flight when G forces act on the body, resulting in a redistribution of the pulmonary capillary blood flow. During exposure to positive ($+G_z$) accelerative forces, the blood flow would be directed to the lung bases, whereas during exposure to negative ($-G_z$) acceleration, the flow would be toward the apical areas.

Composition of Respired Air

The major constituents of atmospheric air are oxygen, nitrogen, and water vapor. The rare gases (argon, krypton, etc.) have not been demonstrated to be biologically significant, and in physiologic gas analysis these concentrations are determined and included in the values reported for nitrogen.

Atmospheric Air.—In dry air at sea level, the partial pressures of the constituent gases according to Dalton's Law are:

$$P_{O_2} = 760 \times 0.2075 = 159.2\text{mm Hg}$$
$$P_{N_2} = 760 \times 0.7902 = 600.6\text{mm Hg}$$
$$P_{CO_2} = 760 \times 0.0003 = 0.2\text{mm Hg}$$

Tracheal Air.—When the inspired air enters the respiratory passages, there is a rapid saturation with water vapor and a simultaneous warming to body temperature. The respiratory organs contain an abundance of water vapor which completely saturates the inspired air, and exerts a constant pressure of 47mm Hg at the nominal body temperature of 98.6° F *regardless of the barometric pressure.* Accordingly, the sum of the partial pressures of the inspired gases no longer equals the barometric pressure, but only the barometric pressure minus the water vapor pressure. Thus, tracheal partial pressure of the inspired gases can be calculated as follows:

$$P_{TR} = (P_B - 47) \times F_I$$

Where

P_{TR} = The tracheal partial pressure of the inspired gas
P_B = Barometric pressure
F_I = The fractional concentration of the inspired gas

Alveolar Air.—The theoretical $P_{O_2(alv)}$ for any altitude can be calculated if one knows the barometric pressure and the dry fraction (percentage) of oxygen in the inhaled gas, if a constant sea level ventilation rate and a normal metabolic rate are presumed for the sake of simplicity. When the $P_{H_2O(tr)}$ is presumed to be a constant 47mm Hg and the $P_{CO_2(alv)}$ to be a constant 40mm Hg, and examination of standard atmosphere tables shows that the barometric pressure at 10,000 feet is 523mm Hg and the dry fraction of O_2 is 21 percent, then at 10,000 feet breathing air,

$$P_{O_2(tr)} = (P_B - P_{H_2O(tr)}) \times .21 \text{ or}$$
$$P_{O_2(tr)} = .21\,(523 - 47) = 99.96\text{mm Hg}$$

However, in the transition from tracheal gas to alveolar gas, the P_{O_2} is reduced, the P_{CO_2} is increased, and the P_{N_2} remains the same.

$$\text{Therefore, } P_{O_2(alv)} = P_{O_2(tr)} - P_{CO_2(alv)} \text{ or}$$
$$P_{O_2(alv)} = 99.96\text{mm Hg} - 40\text{mm Hg} = 60\text{mm Hg}$$

Actual measurements of $P_{O_2(alv)}$ and $P_{CO_2(alv)}$ at various altitudes, both breathing air and breathing 100 percent O_2, are presented in table 11–3. The $P_{O_2(alv)}$ at 10,000 feet was measured at 61mm Hg, so that calculations seen to be in close agreement with the actual figure. However, the $P_{O_2(alv)}$ drop with ascent causes a gradually increasing hypoxic stimulus to respiration (via the chemoreceptors in the area of the carotid sinus) resulting in an increased respiratory exchange rate (RER) and thus an increased $P_{O_2(alv)}$ over that expected, and a decreased $P_{CO_2(alv)}$. Therefore, table 11–3 should be used for precise information and calculations made only when the table and data are not available.

Table 11–4 shows measured changes at sea level in the partial pressure of the gases at various sites in the respiratory cycle.

Figure 11–1 shows the reduction in O_2 tension alone as the inspired O_2 is carried through the vessels to tissue. The partial pressure and concentration of the various alveolar gases are very consistent at sea level.

Oxygen Transport

Oxygen is carried in the blood in simple physical solution and in loose chemical combination with hemoglobin in the form of oxyhemoglobin. The

TABLE 11-3. *Tracheal Oxygen Pressure, Alveolar Oxygen Pressure, and Carbon Dioxide Pressure in the Alveolar Gas When Breathing Air and 100 Percent Oxygen at Physiologically Equivalent Altitudes*

Breathing air						Breathing oxygen (100 percent)				
Altitude	Barometric pressure	Tracheal P_{O_2}	Alveolar		RER*	Altitude	Barometric pressure	Tracheal P_{O_2}	Alveolar	
			P_{O_2}	P_{CO_2}					P_{O_2}	P_{CO_2}
Feet	*mm of Hg*	*mm of Hg*	*mm of Hg*	*mm of Hg*		*Feet*	*mm of Hg*	*mm of Hg*	*mm of Hg*	*mm of Hg*
Sea level	760	149	103	40	0.85	33,000	196	149	109	40
5,000	632	122	79	38	0.87	36,000	170	123	85	38
10,000	523	100	61	36	0.90	39,000	148	100	64	36
15,000	429	80	46	33	0.95	42,000	128	81	48	33
20,000	349	63	33	30	1.00	45,000	111	64	34	30
22,000	321	57	30	28	1.05	46,000	106	59	30	29

*Respiratory exchange rate (Luft, 1961).

TABLE 11–4. *Partial Pressures of Respiratory Gases at Various Sites in Respiratory Circuit of Man at Rest at Sea Level*

Sample	Gas partial pressure				
	O_2 mm Hg	CO_2 mm Hg	N_2 mm Hg	H_2O mm Hg	Total mm Hg
Inspired air	158	0.3	596	5.7	760
Expired air	116	32	565	47	760
Alveolar air	100	40	573	47	760
Arterial blood	100	40	573	47	760
Venous blood	40	46	573	47	706
Tissues	30 or less	50 or more	573	47	700

(Carlson, 1965a.)

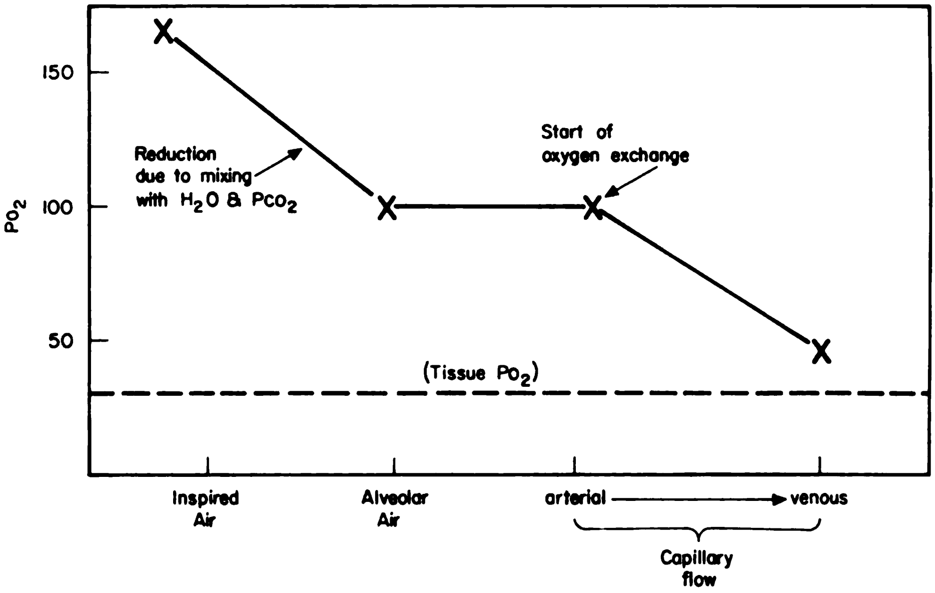

FIGURE 11–1. *Reduction in* P_{O_2} *during respiration.*

oxygen transport capacity of 1 gram of hemoglobin is 1.36ml of oxygen. Therefore, the capacity for 100ml of blood is about 20ml O_2 (presuming normal hemoglobin to be 14.7gm/100cc) representing 100 percent hemoglobin saturation. Normally, as seen in figure 11–2, arterial hemoglobin at sea level, breathing air, is 98 percent saturated. When breathing 100 percent O_2 at sea level pressure, the hemoglobin becomes 100 percent saturated and additional O_2 goes into simple solution in the plasma. The total of additional oxygen so transported is 11 percent greater than normal.

In figure 11-2, the oxyhemoglobin dissociation curves are presented. From the curve, it can be seen that the blood leaves the pulmonary capillaries with the hemoglobin about 98 percent saturated. Even if the $Po_{2(alv)}$ is reduced by 20mm Hg, the saturation is reduced by only three to four percent. In the tissue capillaries, however, a small decrease in oxygen ten-

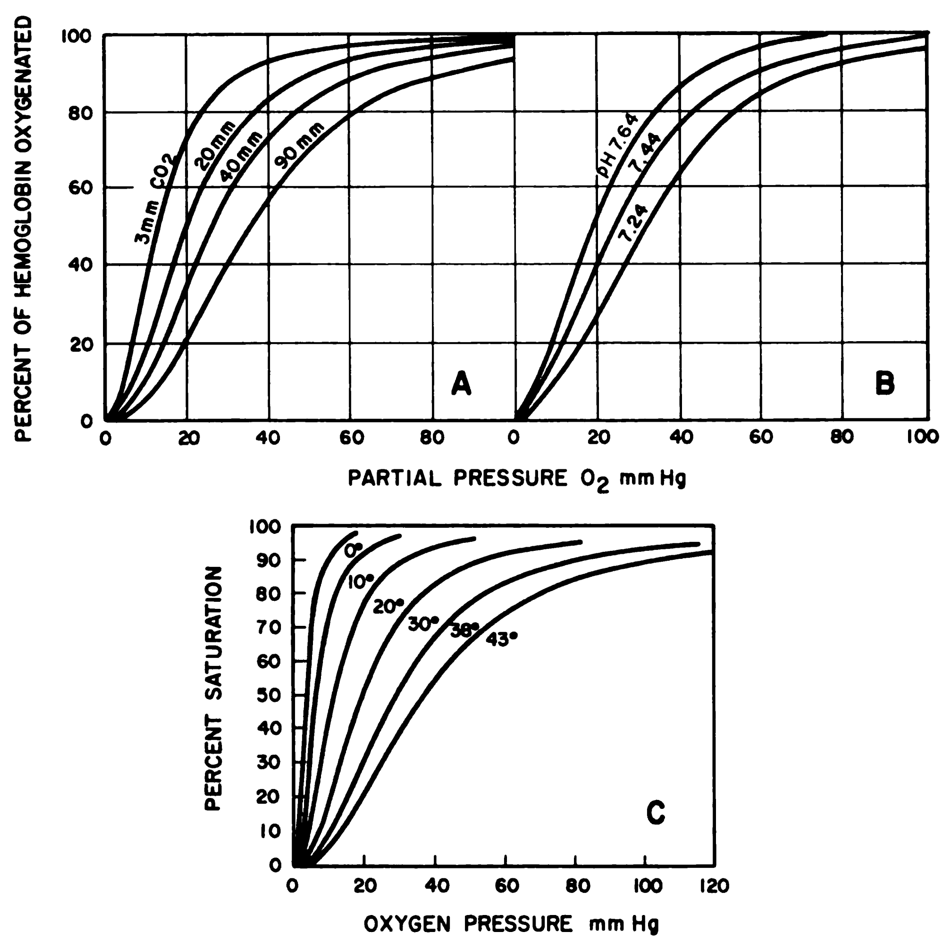

FIGURE 11-2. *A. Effect of* Co_2 *on oxygen dissociation curve of whole blood (after Barcroft). B. Effect of acidity on oxygen dissociation curve of blood (after Peters & Van Slyke). C. Effect of temperature on oxygen dissociation curve of blood* (FROM ROUGHTON, CHAPTER 5 IN Handbook of Respiratory Physiology, W. M. BOOTHBY (ED.), RANDOLPH AIR FORCE BASE, TEXAS, AIR UNIVERSITY, USAF SCHOOL OF AVIATION MEDICINE, 1954). (CARLSON, 1965B.)

sion causes changes in the dissociation curve which result in a large quantity of oxygen being made available to the tissues. The upper section of the dissociation curve remains relatively flat through an oxygen tension change of 40mm Hg; thus, when the $Po_{2(alv)}$ falls from 100 to 60 mm Hg, the blood saturation is reduced approximately eight percent. As the oxygen tension continues to fall, however, the curve illustrates that an additional reduction of 30mm Hg results in a precipitous drop in blood saturation to 58 percent. Thus the characteristic shape of the dissociation curve accounts for the relatively mild effects of hypoxia at low altitude and the very serious impairment at higher altitudes.

The oxygen-carrying capacity of the blood hemoglobin is also very sensitive to changes in blood pH (Bohr Effect) as illustrated in figure 11–2B. At an oxygen tension of 60mm Hg, for example, at pH 7.2, 7.4, and 7.6, the arterial oxygen saturation is observed to be 84, 89, and 94 percent respectively. Carbon dioxide is the major determinant of blood pH. In venous blood, Pco_2 is high; accordingly the pH is low; in arterial blood the Pco_2 is less as a result of the diffusion of CO_2 into the alveoli. The arterial blood, therefore, has a higher pH and can carry more oxygen than would be possible without this change in pH and with the same alveolar Po_2. In the tissues the reverse condition exists.

Control of Respiration

The neural control of respiration is accomplished by neurons in the reticular formation of the medulla. Barcroft (as cited in Ernsting, 1965a) suggested that the basic rhythm of respiration is the result of the inherent activity of the respiratory center and is independent of any reflex excitation from high centers. Recent investigations by Liljestrand (as cited by Ernsting, 1965a) tend to support Barcroft's thesis. This rhythmic activity is modified by afferent impulses arising from receptors in various parts of the body; by impulses originating in higher centers of the central nervous system and by specific local effects resulting from changes in the chemical composition of the blood.

A decrease in the arterial Po_2 causes increased pulmonary ventilation. However, if the afferent fibers from the chemoreceptive areas are severed, respiration is depressed. Thus the direct effect of hypoxia on the respiratory center itself is depressive but hypoxia will cause increased pulmonary ventilation when the chemoreceptor mechanism is intact.

A minute increase of about 0.25 percent alveolar CO_2 will lead to a 100 percent increase in pulmonary ventilation rate. Conversely, lowering the alveolar P_{CO_2} by voluntary hyperventilation tends to produce apnea. From these observations, Haldane (1922) concluded that the control of respiration appears to be governed primarily by the homeostasis of alveolar P_{CO_2}.

Oxygen lack is a rather ineffective stimulus for pulmonary ventilation. Ernsting (1965a) reports that no increase in pulmonary ventilation occurs with *acute* oxygen lack until the alveolar P_{O_2} is reduced to about 65mm Hg, or approximately 37,000 to 39,000 feet equivalent altitude breathing 100 percent O_2, and even a reduction of alveolar oxygen to about 40mm Hg (42,000 feet equivalent altitude) will only increase ventilation by about one-third of its normal resting value. The pattern of pulmonary ventilation occurring in hypoxia does not represent a simple reaction to the reduced alveolar oxygen tension.

An increase in oxygen tension will produce minor changes in respiration. Use of 100 percent oxygen causes an initial slight depression of respiration, followed by moderate stimulation of breathing. The depression is considered to be caused by the effect of high oxygen tension on the chemoreceptors in the aortic and carotid bodies (Gell, 1961). The subsequent stimulation may be caused by the irritant action of 100 percent oxygen on the pulmonary structures.

Under heavy exercise, maximum pulmonary ventilation can reach 110 to 120 liters per minute, as compared to a resting rate of six liters per minute. In aviation, however, extremes of physical activity are not encountered, with the metabolic oxygen consumption seldom exceeding two to three times the resting rate. On this basis, the provision of oxygen in military aircraft is based on a pulmonary ventilation rate of 25 liters per minute.

HYPOXIA

Probably the most frequently encountered lethal hazard in aerospace medicine is hypoxia. Records of early balloon and aircraft flights reveal tragedy from this cause, since even these primitive machines had a higher operational ceiling than the men aboard them.

Hypoxia was a severe problem in both world wars and remains ever present in today's military aviation. Engineering solutions to the problem have been ingenious. Money has been lavished on training, on research and development, and on procurement of equipment to prevent hypoxia. Yet hypoxic incidents continue to occur, and the Flight Surgeon should be extremely well informed of every facet of this problem.

There is a commonly encountered misconception among aviators that it is possible to learn the early symptoms of hypoxia and thereafter take corrective measures when such symptoms are noted. The theory is appealing because it allows all action, both preventive and corrective, to be postponed until the actual occurrence.

Unfortunately, the theory is false and extremely dangerous. One of the earliest effects of hypoxia is impairment of judgment. Therefore, even if the early symptoms are noted, the aviator may disregard them and often does; or he may take corrective action which is actually hazardous, such as disconnecting himself from his only oxygen supply. Finally, at high altitudes, hypoxia may cause complete unconsciousness as the first symptom.

These factors must be kept in mind during the Flight Surgeon's study of hypoxia, during the indoctrination flights in the low pressure chamber at an Aviation Physiology Training Unit, and especially during the Flight Surgeon's daily contact with aviators in the ready room, sickbay, or dispensary.

There are few statistics which tell exactly the number of accidents and fatalities in military aviation which can be attributed to this problem. Some indication of its seriousness, however, can be inferred from an Air Force tabulation over an 18-month period, in which there were 55 recorded hypoxic incidents from which the pilot recovered. These incidents occurred for the most part in training jets which carried two occupants. In many instances, a crash would have resulted almost inevitably had there not been a second pilot aboard to return the aircraft safely to a lower altitude. Obviously, then, many crashes have occurred as a result of hypoxia, although accident statistics do not show this.

A summary report of the Naval Aviation Safety Center (1963) lists accidents and near-accidents in which hypoxia was established or strongly suspected to be the cause. In many of these, the narratives indicate that poor oxygen discipline on the part of the pilot led directly to the occurrence. How many additional instances occur but are corrected prior to an accident, and consequently go unreported, is a matter for conjecture.

Types of Hypoxia

The amount and pressure of oxygen delivered to the tissues is determined by the arterial oxygen saturation, by the total oxygen-carrying capacity, and by the rate of delivery to the tissues. *Hypoxia,* which is defined as an insufficient supply of oxygen, can result from any one of these factors and accordingly, the following different types of hypoxia have been distinguished classically. Other classifications are more precise but also more complex and less useful to the Flight Surgeon.

1. *Hypoxic hypoxia* results from an inadequate oxygenation of the arterial blood and is caused by reduced oxygen partial pressure.
2. *Anemic hypoxia* results from the reduced oxygen carrying capacity of the blood, which may be due to blood loss; any of the anemias; CO poisoning; or by drugs causing methemoglobinemia.
3. *Stagnant hypoxia* is caused by a circulatory malfunction which results, for example, from the venous pooling encountered during acceleration maneuvers.

A fourth type, *histotoxic hypoxia,* is described in the literature as resulting from the inability of the cells to utilize the oxygen provided when the normal oxidation processes have been poisoned by cyanide poisoning. However, as Youmans (1966) points out, there is no oxygen lack in the tissues but rather an inability to use available oxygen, with the result that the Po_2 in the tissues may be higher than normal. Therefore, it is not hypoxia by the definition used here.

The most common type of hypoxia encountered in aviation is hypoxic hypoxia which results from the reduced oxygen partial pressure in the inspired air caused by the decrease in barometric pressure. Other types may also affect aircrew, such as anemic hypoxia (CO poisoning) and stagnant hypoxia resulting from cold or acceleration forces. These are discussed under the toxicology and acceleration chapters respectively.

Types of Onset of Hypoxia

The onset of hypoxia varies with the cause. In the ascent to altitude without supplementary equipment, the onset of hypoxia is as gradual as the rate of ascent. As soon as an inspiration is completed, the alveolar gases approach equilibrium with the inspired gases, and similarly the arterial gases reach a very rapid equilibrium with the alveolar gases, but the change in barometric pressure is gradual between breaths.

In the event of contamination of mask oxygen with some amount of cabin air, due to a leaky mask or tubing, onset is intermittent and the effects inconsistent because the amount of hypoxia varies from one breath to the next, depending on leakage rate, altitude, and body position (which may cause the aperture of a leak to be temporarily closed, partially open, or completely open). This type of hypoxia is baffling to trace because it is difficult to be sure that a hypoxic incident has occurred, much less to determine the cause.

In the case of a supply hose disconnect or other cause of exposure to ambient air, whether known or unknown, the onset of symptoms will be determined by the altitude of the exposure. If such a disconnect is immediately discovered, and if no decompression is involved, one should hold one's breath while attempting to reconnect, because the alveolar Po_2 is higher than ambient Po_2. Breathing in such a circumstance will cause a washout of O_2 from the tissues. This must be avoided until the breath can no longer be held.

When rapid or explosive decompression occurs, the volume and pressure of alveolar gases become markedly higher than those of the ambient atmosphere, and sudden expulsion of the alveolar gases occurs. At the end of the resulting involuntary expiration, the normal reaction is to inhale, and at the end of that inspiration, the alveolar Po_2 is in equilibrium with the ambient. The resulting effects will depend upon the Po_2 at the altitude to which decompressed.

Symptomatology

Many observations have been made on the subjective and objective symptoms of hypoxia which permit a detailed analysis of progressive functional impairment and which indicate that the effects of hypoxia appear to fall into four stages (Air Force Manual 161-1, 1962). Table 11-5 summarizes the stages of hypoxia, the altitude of occurrence, breathing air and breathing 100 percent oxygen, and the arterial oxygen saturation.

TABLE 11-5. *Stages of Hypoxia*

Stage	Altitude in feet		Arterial oxygen saturation (percent)
	Breathing air	Breathing 100 percent O_2	
Indifferent	0-10,000	33,000-39,000	95-90
Compensatory	10,000-15,000	39,000-42,200	90-80
Disturbance	15,000-20,000	42,200-45,200	80-70
Critical	20,000-23,000	45,200-46,800	70-60

Indifferent Stage.—There are no observed impairments and the only adverse effect is on dark-adaptation which serves to emphasize the need for oxygen from the ground up during night flights.

Compensatory Stage.—The physiological adjustments which occur in the respiratory and circulatory systems are adequate to provide defense against the effects of hypoxia. Factors such as environmental stress or prolonged exercise can produce certain decompensations. In general, in this stage, there is an increase in pulse rate, respiratory minute volumes, systolic blood pressure, circulation rate, and cardiac output. There is also an increase in fatigue, irritability, and headache, and a decrease in judgment. The individual has difficulty with simple tasks requiring mental alertness or moderate muscular coordination. Table 11-6 illustrates the physiological compensations which occur over a period of 60 minutes on acute exposure to 18,000 feet.

Disturbance Stage.—In this stage of exposure, the physiological compensations are inadequate to provide sufficient oxygen for the tissues and hypoxia is evident. Subjective symptoms may include headache, fatigue, lassitude, somnolence, dizziness, "air-hunger," and euphoria. At 20,000 feet, the period of useful consciousness is 15 to 20 minutes. In some cases, there are no subjective symptoms noticeable up to the time of unconsciousness. Objective findings include:

1. *Special Senses.* Peripheral and central vision are impaired and visual acuity is diminished. There is weakness and incoordination of the extraocular muscles and reduced range of accommodation. Touch and pain sense are lost. Hearing is one of last senses to be affected.

TABLE 11-6. *Inspired and Alveolar Gas Tensions (mm Hg), and Pulmonary Ventilation at Sea Level, and During an Acute Exposure to 18,000 Feet.*

	Sea level	18,000 feet		
		5 minutes	30 minutes	60 minutes
Barometric pressure (mm Hg)	760	380	380	380
Inspired oxygen tension (tracheal)	150	70	70	70
Alveolar oxygen tension	103	40	38	36
Alveolar carbon dioxide tension	40	33	30.6	30
Respiratory exchange ratio	0.83	1.14	0.95	0.85
Pulmonary ventilation (l./min B.T.P.S.)	8.48	13.51	11.50	10.71
Oxygen tension gradient inspired to alveolar gas (mm Hg)	47	30	32	34

(Ernsting, 1965a, by permission of the Controller of Her Britannic Majesty's Stationery Office.)

2. *Mental Processes.* The most striking symptoms of oxygen deprivation at these altitudes are classed as psychological. These are the ones which make the problem of corrective action so difficult. Intellectual impairment occurs early and the pilot has difficulty recognizing an emergency situation unless he is widely experienced with hypoxia and has been very highly trained. Thinking is slow; memory is faulty; and judgment is poor.

3. *Personality Traits.* In this state of mental disturbance, there may be a release of basic personality traits and emotions. Euphoria, elation, moroseness, pugnaciousness, and gross overconfidence may be manifested. The behavior may appear very similar to that noted in alcoholic intoxication.

4. *Psychomoter Functions.* Muscular coordination is reduced and the performance of fine or delicate muscular movements may be impossible. As a result there is poor handwriting, stammering, and poor coordination in flying.

Hyperventilation is noted.

Cyanosis occurs—most noticeable in the nail beds and lips.

Critical Stage.—In this stage of acute hypoxia, there is almost complete mental and physical incapacitation, resulting in rapid loss of consciousness, convulsions, and finally in failure of respiration, and death.

An important factor in the sequences cited above is the gradual ascent to altitude where the individual can come to equilibrium with the gaseous environment, and physiological adjustments have sufficient time to come into play. Actually this progression occurs in military aviation only in cases where the aviator is unaware that his oxygen is disconnected or in cases where leaks occur in the oxygen system, causing dilution of the oxygen with air.

Of greatest concern to the Flight Surgeon is hypoxia resulting from the sudden loss of cabin pressure in aircraft or vehicles operating at practically unlimited altitudes. Under these conditions, a loss of pressurization or oxygen supply will cause exposure of the aviator to environmental conditions so stressful that physiological compensations cannot occur before the onset of unconsciousness.

Time of Useful Consciousness

The time of useful consciousness is that period of time between an individual's sudden deprivation of oxygen at a given altitude and the onset of physical or mental impairments which prohibit his taking rational action. It represents the time during which the individual can recognize his problem and reestablish an oxygen supply, initiate a descent to lower altitude, or take other corrective action.

The length of time between the loss of oxygen supply and the loss of useful consciousness is determined primarily by the altitude. Other influences such as the inherent tolerance of the individual and the amount of physical activity can affect the time. In healthy young adults at rest, the average times of useful consciousness and the time to unconsciousness at various altitudes are shown in table 11–7 and figures 11–3 and 11–4.

TABLE 11–7. *Time of Useful Consciousness*

Altitude (1,000 feet)	Rapid disconnect (moderate activity)	Rapid disconnect (sitting quietly)
22	5 minutes	10 minutes.
25	2 minutes	3 minutes.
28	1 minute	1 minute 30 seconds.
30	45 seconds	1 minute 15 seconds.
35	30 seconds	45 seconds.
40	18 seconds	30 seconds.
65	12 seconds	12 seconds.

(Carlyle, 1963.)

At higher altitudes, the difference between the time of useful consciousness and that of unconsciousness is very short or nonexistent, since only 12 seconds represents the time of useful consciousness at about 45,000 feet or any higher altitude.

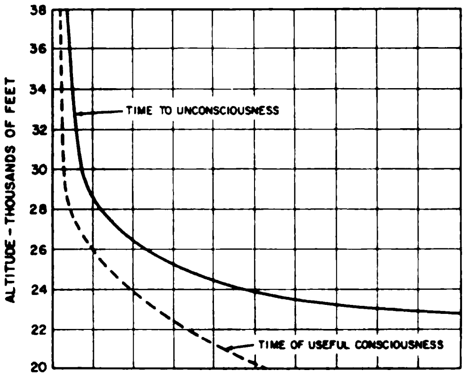

FIGURE 11–3. *The time of useful consciousness and the time to unconsciousness at various altitudes caused by a sudden loss of oxygen supply* (GELL, 1961).

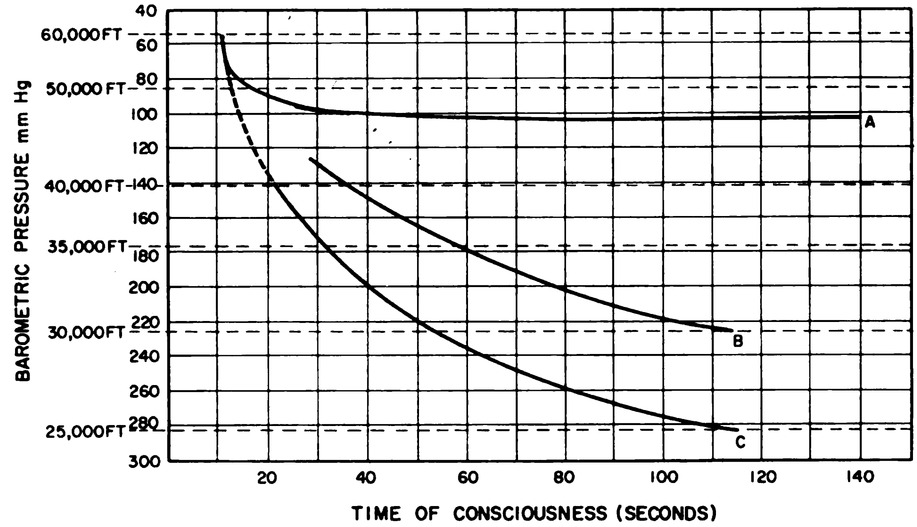

FIGURE 11-4. *Time of consciousness with varying types of exposure at high altitude. A. 100 percent oxygen at ambient pressure—rapid decompression from 40,000 feet* (BENZINGER). *B. Air breathing—mask removal at altitude* (HALL). *C. Air breathing—rapid decompression from 10,000 feet* (WILSON & COMFORT), (APPROACH, JAN. 1965.)

Limit Altitudes and Altitude Equivalents

In considering hypoxia, some sort of limit must be set on the supply of oxygen that would be considered "adequate" for the purposes of military aviation. Ideally, one would select sea level conditions as the limit and design and construct oxygen supply systems to maintain them, but for many reasons this is not feasible for all altitudes at which Navy and Marine Corps aircraft are capable of being operated.

In determining a limit altitude, one is actually specifying the maximum level of hypoxia which is acceptable. In the Navy, NATOPS Manual, *General Flight and Operating Instructions,* OPNAV Instruction 3710.7 series, specifies the following limit altitudes for crewmembers aboard naval aircraft:

1. With one exception, noted below, all occupants aboard naval aircraft will use supplemental oxygen on flights in which the cabin altitude exceeds 10,000 feet. *Exception:* When all occupants are equipped with oxygen, *unpressurized aircraft* may ascend to 35,000 feet. When minimum en route altitudes or an ATC clearance requires flight above 10,000 feet in an unpressurized aircraft, the pilot at the controls shall use oxygen. When oxygen

is not available to other occupants, flight between 10,000–13,000 feet must not be longer than three hours, and flight above 13,000 feet is not authorized.

2. Table 11–8 gives the oxygen requirements for ***pressurized aircraft,*** those flown above 10,000 feet MSL when cabin altitude is maintained at 10,000 feet or less. The quantity of oxygen aboard an aircraft before takeoff must be sufficient to accomplish the planned mission. In aircraft carrying passengers, there must be an adequate quantity of oxygen to protect all occupants through normal descent to 10,000 feet.

TABLE 11–8. *Oxygen Requirements for Pressurized Aircraft*

Ambient altitude	One pilot	Second pilot	Crew on duty	Other occupants
Below FL 270	R	R	R	N/A
FL 270 through FL 350	I	R	R	R
Above FL 350 through FL 400	I or O	I R	R	R
Above FL 400 through FL 450	O	I	R	R
Above FL 450 through FL 500	O	I	I	I
Above FL 500	P	P	P	P

Legend:

R—Oxygen shall be readily available.

I—Oxygen shall be immediately available. Helmets shall be worn with an oxygen mask attached to one side or an approved quick-donning or sweep-on mask properly adjusted and positioned for immediate use. Set oxygen regulator to 100 percent and ON.

O—Oxygen shall be used.

P—Pressure suit shall be worn.

(OPNAVINST 3710.7 series)

If loss of pressurization occurs, a descent shall be made immediately to a flight level where cabin altitude can be maintained at or below 35,000 feet and oxygen shall be utilized by all occupants. (This restriction does not apply when a functioning pressure suit is worn.)

When it is observed or suspected that an occupant of any aircraft is suffering the effects of decompression sickness, the pilot will immediately descend, land at the nearest installation, and obtain qualified medical assistance. The person affected may continue the flight only on the advice of a Flight Surgeon.

3. In *pressurized combat and combat training jet aircraft,* oxygen shall be used by all crewmembers from takeoff to landing. Emergency bailout bottles, where provided, shall be connected prior to takeoff. Whenever practicable, 100 percent oxygen shall be used for takeoff and landing. Flights above 50,000 feet may not be made without Naval Air Systems Command authorization.

The most important measure physiologically at any altitude is the alveolar partial pressure of oxygen, or $Po_{2(alv)}$, since it is essentially this value in the individual at rest under normal ambient gas conditions (i.e., in the absence of toxic gases) which determines arterial O_2 saturation and thus tissue oxygenation. Therefore, for practical purposes, one may consider that any altitude (or barometric pressure) may be expressed in terms of a $Po_{2(alv)}$, either while breathing air or while breathing 100 percent O_2. For the purpose of establishing limit altitudes then, the $Po_{2(alv)}$ will be the deciding factor.

Respiratory Adjustments to Altitude

The critical $Po_{2(alv)}$ *at which the average individual loses consciousness on short exposure to altitude is 30mm Hg.* This corresponds to 23,000 to 25,000 feet on curve A of figure 11–5. In the complete absence of respiratory adjustments to altitude, the same $Po_{2(alv)}$ would be encountered at about 17,000 feet. Applying similar considerations to 100 percent O_2 breathing altitudes, it is evident that hypoxia-induced hyperventilation, as reflected in the course of the $Pco_{2(alv)}$ on curve D of figure 11–5, does improve the $Po_{2(alv)}$ measurably. Thus the 30mm $Po_{2(alv)}$ is noted at 47,000 feet with respiratory adjustment and 44,000 feet without it.

Comparisons can be made between different barometic pressures which produce the same alveolar Po_2 when breathing air in one case and 100 percent O_2 in the other in order to establish "physiologically equivalent altitudes." Actually, physiological states cannot be compared solely on the basis of $Po_{2(alv)}$ but must consider $Pco_{2(alv)}$ and ventilation also, since an imbalance in any one will cause change in the others until a steady state is reached.

The time necessary to reach a steady state at various altitudes is given in figure 11–6. Note that even at the relatively low altitude of 18,000 feet a steady state is reached only after an hour of respiratory adjustment. Such considerations, however, need not concern the practicing Flight Surgeon unduly. For practical purposes, the $Po_{2(alv)}$ may be used without considering respiratory adjustment in establishing physiologically equivalent altitudes.

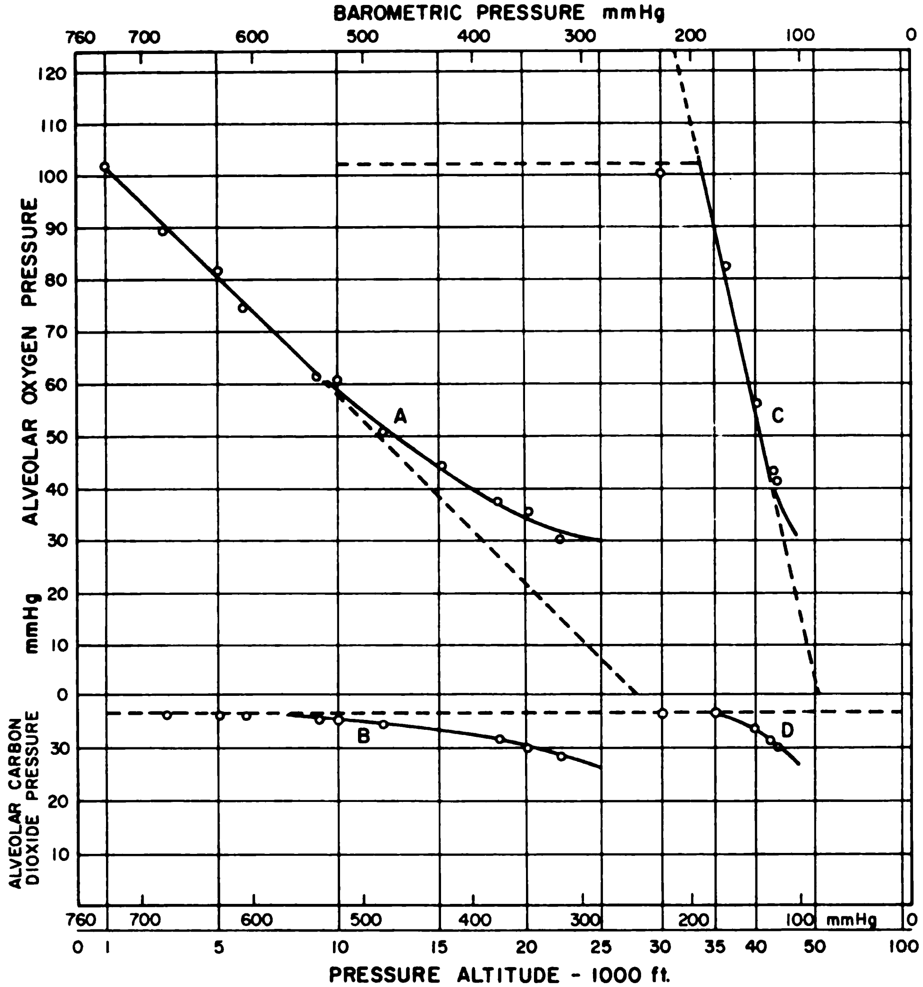

Figure 11–5. *The partial pressures of respiratory gases when breathing air (A, oxygen; B, carbon dioxide) and using oxygen equipment (C, oxygen; D, carbon dioxide). The interrupted lines represent the theoretical course in the absence of the respiratory response to hypoxia at altitude* (BOOTHBY, LOVELACE, BENSON, & STREHLER, 1954).

Ten thousand feet during daylight is specified as the limit above which, in nonpressurized aircraft, all crewmembers must use oxygen. The $Po_{2(alv)}$ at 10,000 feet, breathing air, is approximately 61mm Hg, which is the maximum acceptable level of hypoxia which Marine Corps and Navy aircrewmen are allowed to undergo. As a consequence, all oxygen equipment and barometric controls are designed to maintain the user at this physiological equivalent or below.

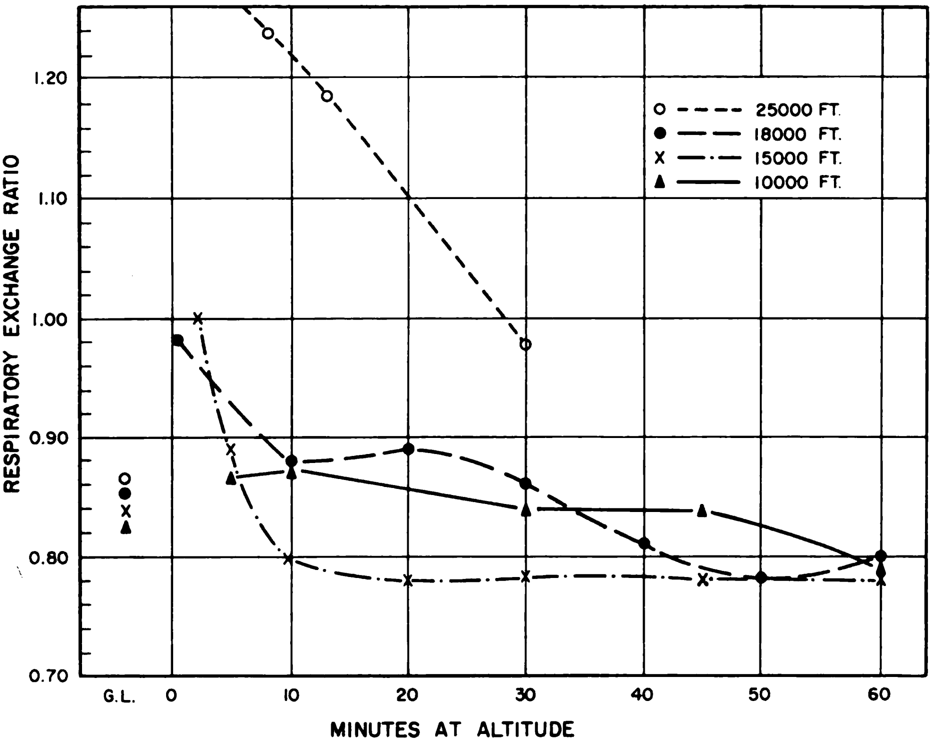

Figure 11–6. *The respiratory exchange ratio in the course of exposures to 10,000, 15,000, 18,000 and 25,000 feet, indicating the duration of the "unsteady state"* (BOOTHBY ET AL., 1954).

Having arrived at the lower limit of $Po_{2(alv)}$ allowable in Navy and Marine Corps aviation, one may now compare various equivalent altitudes yielding the same $Po_{2(alv)}$. In breathing O_2 *not* under pressure, table 11–9 shows a $Po_{2(alv)}$ of 61mm Hg at 39,500 feet which is therefore the upper limit for flying without positive pressure breathing. Similarly, other limiting altitudes are noted in table 11–9.

A question may arise as to why 10,000 feet, breathing air, or a $Po_{2(alv)}$ about 60mm Hg was selected as the upper limit. Reference to table 11–5 shows that 10,000 feet breathing air is the upper limit of the indifferent stage of hypoxia. Even more important, reference to the oxyhemoglobin saturation curve shows that the ascent to 10,000 feet caused a decrease of only about seven percent in the oxyhemoglobin saturation, since at 10,000 feet the hemoglobin is still 90 percent saturated. However, rather small increases in altitude thereafter cause a rather marked steepening of the slope of the curve.

Certainly a 2- to 3,000-foot difference won't matter too much, but anything over that becomes unacceptable—hence the NATOPS limitation to 13,000 feet for not over three hours.

The theoretical considerations just mentioned set limits which are useful in making predictions and calculations. In military usage, however, many

TABLE 11–9. *Limiting Altitudes for Respiratory Functioning*

Altitude	Atmospheric pressure (mm Hg)	Alveolar Po_2 (mm Hg)		Reaction	Protection
		Breathing air	Breathing 100 percent O_2		
63,000	47			In theory, body water vaporizes at this altitude or above.	
50,000	87			Effective limit for short-time mask pressure breathing.	Full pressure suit mandatory for any higher altitude.
43,000	122		44	Effective limit of pressure breathing for sustained flight.	Full pressure suit mandatory for extended exposure above this altitude.
39,500	144		61	$O_{2(alv)}$ drops to level equal to 10,000 feet even with 100 percent O_2.	Limit for nonpressure breathing masks.
35,000	179		90		Maximum altitude maintained by full pressure suit.
33,700	191		103	$O_{2(alv)}$ with 100 percent O_2 equal to that at sea level breathing air.	Regulators begin positive pressure O_2 delivery.
27,000	259	27	180		Diluter demand O_2 system goes to 100 percent O_2.
18,500	372	37	300	Usual lower limit for possible occurrence of dysbarism.	
10,000	523	61	443	Lower limit for compensatory stage of hypoxia.	O_2 used on all flights above 10,000 feet.
5,000	632	79	550	Lower limit for hypoxic effects on special senses.	O_2 is recommended above 5,000 feet on night flights.
Sea level	760	103	673		

variable factors must be taken into account. If the oxygen mask suspension is not tightly adjusted or is a bad fit for the individual aviator, a lower $Po_{2(alv)}$ will be produced in the individual than the equipment settings theoretically would produce, due to dilution of the inspired oxygen with cabin air. Many similar factors could account for considerable variation in the absolute Po_2 delivered to the trachea in the same individual at the same altitude with the same equipment at the same settings, but on different days or even different flights.

Differences between individuals in diffusion rates for the alveolar membrane, or in the amount of circulating hemoglobin, or in several other variables, could also result in a lower arterial Po_2 than expected from the same $Po_{2(alv)}$.

Pierce, Lambertsen, Strong, Alexander, and Steele (1962) obtained precise measurements of the changes in respiratory gas pressures with a change from sea level (760mm Hg) to a simulated altitude of 39,000 feet (147mm Hg). At 39,000 feet, breathing 100 percent O_2, subjects developed relative hypocapnia and moderate hypoxia (table 11–10).

TABLE 11–10. *Changes in Respiratory Gas Pressures at a Simulated Altitude of 39,000 Feet*

	Sea level	39,000 feet
Arterial Po_2	103mm Hg	56mm Hg.
Arterial Pco_2	43.5	40.3.
Arterial hemoglobin saturation	96.9 percent	87.1 percent.
Mean brain capillary Po_2	54	40.
Cerebral venous Po_2	37	29.
Cerebral venous hemoglobin saturation	64.4 percent	52.9 percent.

The development of hypoxia is reflected not only in the lowering of Po_2 in the arterial blood, but also in the brain capillaries and venous blood.

Comparison of Pierce's data with the data for the same situation in table 11–3 shows considerable variance. The significance to aerospace medicine is that the wide range of variability both in supply and among individuals must be compensated for in some manner by the supply of oxygen. The mechanical means will be discussed later, but one concrete example of the built-in safety factors in oxygen equipment is given here.

From calculations of $P_{O_2(alv)}$, as noted in table 11–9, 33,700 feet is the altitude at which the individual breathing 100 percent oxygen has the same $P_{O_2(alv)}$ as the same individual breathing air at sea level. If no safety factor were included, the aneroid of the diluter-demand oxygen regulator would be set so that the regulator would deliver 100 percent oxygen at that altitude, since oxygen would be wasted if the regulator were set to deliver 100 percent at any lower altitude. (The reason for attempting to conserve oxygen is that it is a limiting factor on aircraft range.)

In actuality, the aneroid on the diluter-demand regulator is set to deliver 100 percent oxygen at 27,000 feet rather than 33,700. Such safety factors are built into almost all Navy lifesaving equipment, not only to cover the range of human variation but also the possibilities of some slight misuse or maladjustment of the equipment.

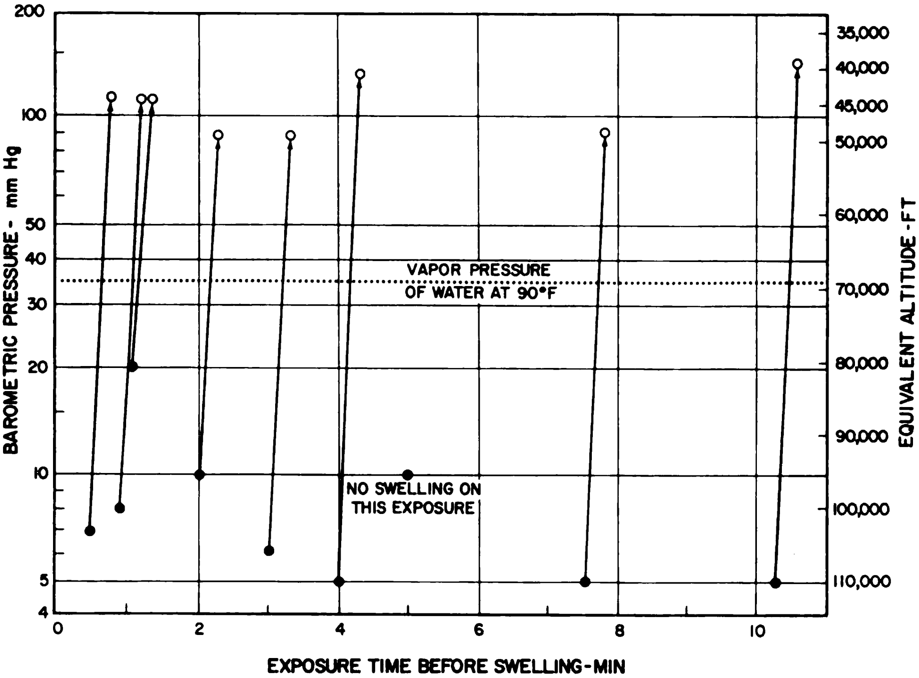

FIGURE 11–7. *Water vapor in tissue at extreme altitudes.* (BILLINGS & ROTH, 1964; DATA FROM WILSON).

The theoretical upper limit of altitude which can be endured by the unprotected body is the point at which the ambient pressure is equal to or lower than the vapor pressure of water at body temperature of 98.6° F, in which case all the water in the body would vaporize. This would occur at 63,000 feet with a barometric pressure of 47mm Hg. Actually this "critical" altitude must be modified upward, since the water in the body is contained in the pressure vessels of cells, intravascular spaces, etc. The only situation in which it might occur is one in which an aviator is flying at or above this altitude (with the cabin pressurized to a much lower altitude) and experiences a rapid decompression to ambient pressure.

This upper limit has been tested experimentally and appears to be rather on the low side of the actual figure.

In experiments on the unprotected human hand (figure 11–7, Billings & Roth, 1964), it was found that a pressure below that equal to water vapor pressure at skin temperature was required to cause vaporization of body water. The discrepancy may have been due to the forces exerted by connective tissues within the hand, and the elastic nature of the skin covering.

Appearance of water vapor occurred suddenly and manifested itself by marked swelling of the hand after a variable time at altitude. After appearance of swelling, the pressure in the altitude chamber was quickly raised; the hand was examined periodically. The upper point (o) represents the first point at which swelling was no longer visible to the eye. (X-ray photographs showed some residual vapor at somewhat higher pressures in a few experiments.)

If chamber pressure was again lowered slightly, swelling again appeared, indicating the continued presence of bubble nuclei in the hand tissues. This suggests that once water vapor bubbles appear, O_2 and CO_2 diffuse into the bubbles, which thus come to be made up of gas saturated with water vapor.

For the Navy and Marine Corps aviator, however, the NATOPS Manual, OPNAVINST 3710.7 series limits flights in *pressurized* aircraft flown by aviators *not* utilizing full pressure suits to 50,000 feet (again, that built-in safety factor!). Aviators and aircrew equipped with pressure suits and in pressurized aircraft—*but only with Naval Air Systems Command authorization,* may exceed 50,000 feet ambient altitude. The Flight Surgeon has a definite responsibility in the indoctrination and continuing education of flight personnel in the medical and physiological factors inherent in use of pressure suits. Since the MK IV full pressure suit could be worn safely on the surface of the moon if an appropriate life support system were furnished to solve other problems, the wearing of the full pressure suit would appear to afford the aviator unlimited utilization of the altitude capabilities of his aircraft.

Positive Pressure Breathing

When breathing 100 percent O_2, the altitude limit of $P_{O_2(alv)}$ of about 60mm Hg is reached at 39,000 feet. Since military aviation exists to win battles and since even in today's air warfare an altitude advantage may be decisive, some means must be found to increase the aviator's ceiling. The only way in which this can be done is to apply positive pressure to the 100 percent O_2 inhaled by the aviator. In its simplest form, this pressure is applied by a positive pressure regulator to a pressure breathing mask, thus increasing the $P_{O_2(alv)}$ by the amount of the pressure applied in excess of ambient. However, the increased intra-alveolar pressure also may cause serious side effects which depend upon the amount of pressure applied.

For the interested Flight Surgeon, an excellent review of the physiology of pressure breathing, by Ernsting (1965c) is available in Gillies' *Textbook of Aviation Physiology*. For purposes of this discussion, however, a short summary will suffice. To understand the mechanism of pressure breathing, consider the following example.

At 39,000 feet, breathing 100 percent O_2, the $P_{O_2(alv)}$ is about 64mm Hg, the Navy's limiting altitude, as previously noted. As the ascent continues, the regulator increases the pressure at which the O_2 is delivered so as to maintain the $P_{O_2(alv)}$ of about 60mm Hg. At 43,000 feet, the regulator is delivering about 15mm Hg positive pressure. At this altitude, P_B is 122mm Hg, $P_{H_2O(alv)}$ is 47mm Hg, and table 11-3 shows that $P_{CO_2(alv)}$ is about 32mm Hg. Therefore, without the positive pressure, the regulator would be delivering 122 minus (47+32) or 43mm Hg $P_{O_2(alv)}$. This is roughly equivalent to 15,000 feet breathing air. With the added 15mm Hg positive pressure, however, the $P_{O_2(alv)}$ is 43+15 or 58mm Hg, again very close to the limit altitude.

Fifteen mm Hg represents a practical maximum for sustained positive pressure, in view of the side effects. The major side effects (due entirely to increased intrapulmonary pressure) are decreased venous return to the heart with consequent decreased cardiac output, increased arterial blood pressure, distention of extrathoracic veins, tachycardia, and possible rupture of alveoli. Alveolar rupture has been shown to occur in animals at pressures of 60mm Hg with an open thorax or at about 100mm Hg with a normal chest. The decreased cardiac output plus the tachycardia results in a marked decrease in stroke volume. Furthermore, Ernsting states that positive pressure breathing leads to syncope but the length of time which elapses before onset of syncope depends primarily on the pressure applied. At a positive pressure of 20mm Hg, for example, most subjects can pressure breathe for 30 minutes without syncope.

The advantages of a positive pressure mask are:

1. The equipment is inexpensive, reliable, instantly available, requires comparatively little maintenance over that normally required and presents a small target.
2. With a small amount of training, a definite increase in service ceiling can be obtained.

The disadvantages are:

1. The service ceiling increase is small (about 5,000 feet) and limited.
2. The limitations are those caused by possible injury to the aviator.
3. Pressure breathing is opposite to the normal breathing pattern in that inhalation is passive and exhalation active, thus requiring training and familiarization.
4. The process of pressure breathing is fatiguing.
5. Communications are much more difficult during pressure breathing.
6. Hyperventilation with resulting respiratory hypocapnia is very common even in moderately experienced aviators.

The use of pressure breathing in naval aircraft is almost exclusively reserved for emergencies, such as penetration or disruption of the aircraft pressure cabin. However, the A-13A mask and all regulators in general use provide this modality and the aviator must be prepared to utilize it if necessary.

Bailout Oxygen Supply and Special Breathing Techniques

All first-line jet aircraft have an emergency oxygen supply in a high pressure (1,800 psi) oxygen bottle contained in the seat kit for the ejection seat. There is a different capacity bottle for each seat-aircraft combination so that oxygen durations for a given flow rate cannot be calculated. The range of capacities is from about 47 cubic inches in the A-4 to about 103 cubic inches in the F-4 aircraft.

The Chief of Naval Operations has recently promulgated a requirement for future emergency oxygen systems which states that the capacity must be sufficient to provide oxygen to an aviator following an ejection at 60,000 feet for the entire duration of descent, plus an additional five minutes at sea level. The amount required would be about 120 cubic inches with present regulators.

The bailout bottle is automatically actuated by ejection and provides oxygen through the minireg and A–13A mask during descent.

Time to Ground.—Bailout oxygen is necessary because of the considerable time durations in descent by free fall from high altitude, and even longer times when the parachute is opened prematurely (see chapter on Emergency Escape from Aircraft). Table 11–11 shows that time of useful consciousness at 75,000 feet is 12 seconds, while time to fall to 14,000 feet (an arbitrary altitude at which consciousness can be maintained) in free fall is 150 seconds, and with a 28- to 30-foot parachute open, is 1,680 seconds (28 minutes). From 40,000 feet, time of useful consciousness is 18 seconds while time to free fall to 14,000 feet is 90 seconds, and time to descend to 14,000 feet is 900 seconds (or 15 minutes) with the 28- to 30-foot parachute open (Webster, 1947).

TABLE 11–11. *Period of Useful Consciousness in High Altitude Bailout*

Bailout altitude (feet)	(Time in seconds)		
	Useful consciousness	Free fall to 14,000 (feet)	28- to 30-foot chute to 14,000 feet
75,000	12	150	1,680 (28 minutes).
55,000	12	120	1,200 (20 minutes).
40,000	18	90	About 900 (15 minutes).
30,000	75	60	600 (10 minutes).

Obviously, some provision must be made to keep the pilot alive during such a parachute descent or conscious during free fall. Barometrically actuated parachute openers allow the aviator to free fall in the unconscious condition and survive, but accidental parachute deployment at high altitude would cause certain death or at least unconsciousness from hypoxia if emergency oxygen could not be supplied. Note that in figure 11–8 the time to free fall from 28,000 feet to 14,000 feet is the same as the useful consciousness time at 28,000 feet. For rough approximations, therefore, 28,000 feet is the highest altitude from which free fall can be accomplished while breathing ambient air and retaining consciousness. Actually, the time of useful consciousness increases as the subject falls but this may be considered a safety factor.

Emergency Breathing Techniques.—In earlier times, several techniques were developed for increasing the pressure of oxygen in the alveoli by alteration of breathing. These are:

1. *Hyperventilation:* covered elsewhere.
2. *"Grunt-breathing":* where a deep breath is taken, the glottis is closed, a hard grunt is made, then the breath is released; repeating the entire process as often as necessary.
3. *"Peak-breathing":* where a deep breath is taken and released while repeatedly shouting the word "PEAK" with short, rapid exhalation effort; again repeating as often as necessary.

Such homegrown methods of pressure breathing may give sufficient $P_{O_2(alv)}$ increase to allow retention of consciousness during free fall from high altitudes. Generally speaking, such methods are no longer necessary due to adoption of the minireg, which allows regulated breathing of emergency oxygen during descent, but may be utilized for descent when the minireg is not installed.

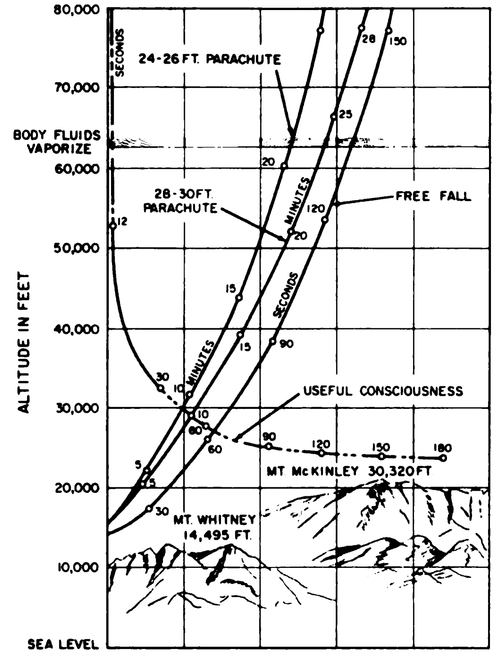

FIGURE 11–8. *Descent to safe breathing altitude* (CARLYLE, 1963).

Acclimatization to Altitude

Acclimatization to altitude is primarily of importance for aviators when suddenly required to begin day-to-day ground operations at altitudes in excess of 15,000 feet. The definitive work on the exact chronology of all of the biochemical and physiological mechanisms remains to be done. An excellent summary of present knowledge is contained in Lambertsen's (1961) article in Bard's *Medical Physiology.* Gell (1961) found that the most accurate measure of complete acclimatization is the increase in hemoglobin in the blood to a degree which assures the same oxygen transport capacity of the blood as at sea level and a heart rate which is equal to or less than that at sea level, both at rest and with exercise.

Considerable research has been conducted concerning acclimatization effects among the Peruvian Indians of Morococha, who spend their entire lives at altitudes above 10,000 feet. They apparently have adjusted completely to the low ambient pressure and can even engage in strenuous physical activity with no ill effects. That this acclimatization is genuine is shown by the fact that these individuals demonstrate a much greater duration of useful consciousness time when exposed suddenly to great simulated heights in a low pressure chamber. For example, during a test at 30,000 feet, half of the exposed natives retained full consciousness and were able to write for an indefinite time (Velasquez, 1959).

One of the principal adaptive mechanisms of natural acclimatization is a lessening of the Po_2 gradient from inspired air to mixed venous blood as shown in figure 11–9 (Hurtado, 1964). It is apparent that, for the Morocochan residents, mechanisms for the transfer of oxygen within the body have developed to a remarkable degree of efficiency.

Another change noted in high altitude acclimatization is an increase in pulmonary ventilation. In the Morococha residents, the ventilation was found to be about 20 percent greater than at sea level, and this percentage rises to about 40 percent if related to body weight or to body surface area. Although a number of factors have been suggested as possibly responsible for this high altitude hyperventilation, the exact reasons are not completely understood.

Although cardiac output is little changed in the acclimatized individual, there are a number of other changes related to the vascular system. It has been demonstrated repeatedly (Hurtado, 1964) that prolonged or permanent exposure to high altitude produces polycythemia. Residents of Morococha,

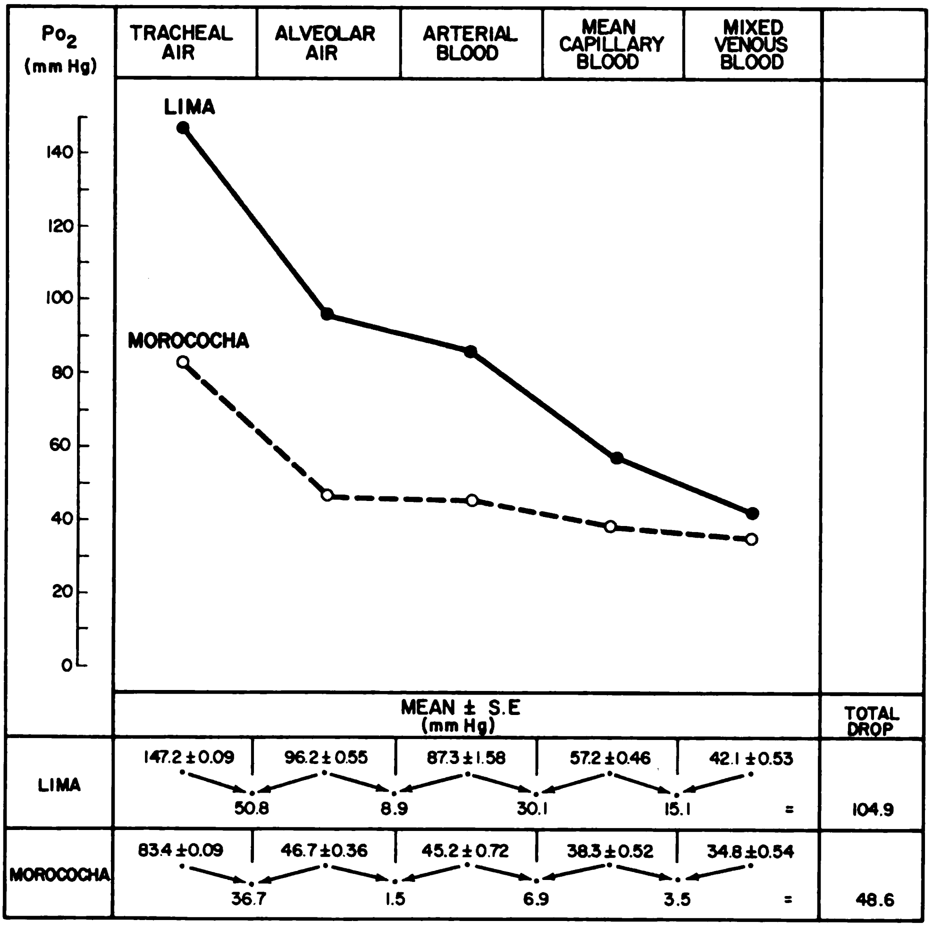

FIGURE 11–9. *Mean Po_2 pressure gradients, from tracheal air to mixed venous blood, in native residents of Lima (sea level) and Morococha (4,540 m). Mean values correspond to two groups of eight healthy adult subjects each, studied at sea level and at high altitudes. Alveolar air, arterial blood, and mixed venous blood (from the pulmonary artery) obtained simultaneously, at rest in the recumbent position. Mean capillary blood Po_2 calculated* (HURTADO, 1964).

for example, were found to have a significant increase in the number of red blood cells and amount of hemoglobin and in the hematocrit, but normal sea level values for leucocytes and platelets. These last characteristics confirmed that a hypoxic condition constitutes, so far as blood is concerned, a specific stimulus only for erythropoiesis. Additionally, it was found that there was a tremendously increased vascularization within the bodies of animals living at the high altitudes. There was a significant increase in the number of capillaries within any section of tissue. Although such quantitative determinations have not been made in humans, indirect observations seem to indicate that a similar condition exists. The increased capillary bed is an important adaptive mechanism inasmuch as it greatly favors the diffusion of oxygen from blood to tissues.

Van Liere and Stickney (1963) summarize several studies of possible acclimatization made of aviators. In general, these studies show minimal changes, if any at all. In one study, some evidence was found for heightened erythropoiesis, but the authors concluded that on the whole there was no evidence that fliers developed compensatory adjustments to a constant and significant degree. Inasmuch as alveolar oxygen tensions for aviators are maintained, through use of oxygen equipment, at levels equivalent to 10,000 feet or less, the appearance of major acclimatization effects would be rather surprising.

Hyperventilation

Hyperventilation is caused by increases in depth and/or frequency of breathing. The cause for the increase may be physiological, as in the case of increased $P_{CO_2(art)}$ due to breath holding, or it may be "voluntary" or "active," as in the case of the experimental subject who is asked to hyperventilate. Another "voluntary" cause is anxiety or fear, although the individual is usually unaware that he is hyperventilating.

The physiological stimuli of hyperventilation are blood CO_2 buildup, or, to a far lesser extent, blood O_2 decrease. In gradual ascents to altitude without supplemental O_2, significant hyperventilation due to decreased $P_{O_2(art)}$ usually does not occur below 10,000 feet. As ascent continues beyond 10,000 feet, the hyperventilation gradually increases. Voluntary or anxiety-induced hyperventilation can, and frequently does, occur at sea level, but may occur under any set of circumstances.

The net result of hyperventilation, regardless of cause, is hypocapnia along with varying effects on blood oxygenation, which will be further detailed. The hypocapnia may be of such severity that tetany may occur. If the cause is hypoxia, a mixture of the symptoms due to hypoxia and those due to hyperventilation may be present. The differentiation of symptoms due specifically to each cause is difficult.

The ***hyperventilation syndrome*** results from hyperventilation in the presence of normal $P_{O_2(alv)}$ and is common in the general population as well as in aviation personnel, both in the air and on the ground. It is usually associated with anxiety or excitement and frequently occurs during emergency situations. Conversely, the hyperventilation syndrome may precipitate an emergency when it develops in an aviator in flight or in a low pressure chamber indoctrination.

The typical symptoms are: a feeling of apprehension; dizziness and weakness; numbness and tingling of the face, fingers, and toes; neuromuscular irritability with fasciculation and tetany, mental confusion of a severe nature, and possible syncope. The most prominent sign is increased respiratory rate and/or depth.

Since many of the same symptoms occur with hypoxia, how can one make an immediate differential diagnosis on the scene—such as over the radio? Such an undertaking is difficult because of the interdependent nature of the two conditions, but if it is known that oxygen is being supplied, and that the major complaints are numbness and tingling of the hands, face, and feet, then hyperventilation can be postulated. If the incident occurs at 10,000 feet or below and the aviator is breathing ambient air, hyperventilation is the presumptive diagnosis. If the incident occurs at altitude, however, and if the Flight Surgeon is consulted by radio, the differential diagnosis must be made in a hurry. The obvious solution is to recommend descent to 10,000 feet or below as rapidly as possible and then reevaluate. While descending, the pilot must be warned not to disconnect the oxygen supply when hypoxia or hyperventilation is suspected.

All aviators should be familiar with the symptoms of the hyperventilation syndrome, and should be trained to take the necessary corrective action, which is simply breath holding.

Hyperventilation Effects on Oxygen Levels.—The effect of respiratory compensatory mechanisms on $P_{O_2(alv)}$ during exposure to decreased O_2 partial pressures is shown in figure 11–5. The mechanisms are somewhat complex.

Hypoxia causes hyperventilation when the $P_{O_2(alv)}$ drops to a sufficiently low level. This results in hypocapnia in a matter of seconds, as CO_2 is blown off, which in turn causes an increase in blood pH, as noted in table 11–12. It also results in an increased $P_{O_2(art)}$, but the alkalosis as well as the decreased $P_{O_2(art)}$ shifts the oxyhemoglobin dissociation curve to the left (Bohr effect), as noted in figure 11–2, causing relatively less oxygen to be made available to the tissues.

The alkalosis also produces brain arterial vasoconstriction. This effect was beautifully demonstrated by Pierce et al. (1962). In a study conducted at

39,000 feet, they found that, as shown in table 11–13, hyperventilation while breathing 100 percent O_2 increased $P_{O_2(art)}$ markedly, but the resulting hypocapnia decreased cerebral blood flow by almost 50 percent, resulting in a net decrease in brain oxygenation as compared with normal breathing of 100 percent O_2 at that altitude. On the other hand, breathing normally at the same altitude, on a 30 percent CO_2–70 percent O_2 mixture *increased* brain oxygenation slightly over normal breathing of 100 percent O_2 despite lower $P_{O_2(art)}$ values, because the increased $P_{CO_2(art)}$ caused cerebral vasodilation which restored cerebral blood flow to sea level values. This effect is limited, however, to altitudes below 40,000 feet and above 35,000 feet.

TABLE 11–12. *Effect of Normal Increased and Decreased Alveolar Ventilation on Arterial Blood*

Type of ventilation (Resting man)	Alveolar ventilation, L/min	Alveolar and arterial gas tensions*		Arterial blood gas contents		Arterial pH units
		O_2 mm Hg	CO_2 mm Hg	O_2 percent saturated	CO_2 mM/L	
Hypoventilation	2.50	67	69	88.5	27.2	7.24
Normal	4.27	104	40	97.4	21.9	7.40
Hyperventilation	7.50	122	23	98.8	17.5	7.56

*In each case, respiratory exchange ratio of 0.8 and O_2 consumption of 250 ml/min are assumed, and the values are calculated from the CO_2—O_2 diagram of Rahn and Fenn. (Bryan, 1964; sources: Comroe et al., 1962; Rahn & Fenn, 1955).

TABLE 11–13. *Effect of Hyperventilation on Respiratory Gas Pressures at a Simulated Altitude of 39,000 Feet, Breathing 100 Percent O_2*

	Normal respiration 39,000 feet	Hyperventilation 39,000 feet
Arterial P_{O_2}	55mm Hg	81mm Hg.
Arterial P_{CO_2}	40.3	21.7.
Arterial hemoglobin saturation	87.1 percent	96.9 percent.
Mean brain capillary P_{O_2}	39.5	35.1.
Cerebral venous P_{O_2}	29	20.
Cerebral venous hemoglobin saturation	52.9 percent	37.7 percent.
Respiration rate	12.4/minute	33.8/minute.

Oxygen Paradox

The restoration of the normal alveolar oxygen tension in the hypoxic subject can be accompanied by a temporary increase in severity of the symptoms. This condition is referred to as the "oxygen paradox," and it is generally found to occur with sudden oxygen administration and reoxygenation. Spasms and unconsciousness can occur during the episode, which may last from a few seconds to several minutes.

The hypocapnia accompanying hypoxia as well as the fall in arterial blood pressure, which has been demonstrated by many investigators, may work in concert to cause a reduction of cerebral blood flow following reoxygenation of the blood. This reduced blood flow would serve to intensify the cerebral hypoxia for a brief period while the carbon dioxide tension of the brain tissue returns to its normal level.

In other cases, respiration is markedly depressed by administering O_2 when the patient has been hypoxic for some time, because hyperventilation associated with the hypoxia has combined with the decreased supply of tissue oxygen, to cause hypocapnia of such a degree that the CO_2 stimulus to respiration is removed. In such a case, the hypoxic stimulus is the only one which remains, and it is removed when pure O_2 is supplied, causing sudden onset of apnea, with gradual return to normal respiration as the CO_2 builds up.

In still other cases, Luft (1954) found that sudden exposure of an individual at 33,000 feet on 100 percent O_2 to ambient P_{O_2} at altitudes above 52,000 feet caused almost instant hypoxia with O_2 saturation in the ear remaining almost unchanged for four to five seconds (giving a measure of lung-brain circulation time). Thereafter, O_2 saturation dropped rapidly, reaching a minimum in nine to ten seconds. At exactly 16 seconds, unconsciousness set in abruptly. In succeeding tests, the time of exposure was successively reduced from 16 to 12 to 8 seconds in order to determine the minimal exposure necessary to cause unconsciousness. The subjects became unconscious in all tests in which they were exposed for over six seconds, and unconsciousness regularly occurred only after 15 to 16 seconds, despite the fact that the subject was again breathing 100 percent O_2 by that time. This delayed action of hypoxia was absent only when exposure to the hypoxic stimulus was less than six seconds. Luft states that the latency of the anoxic symptoms cannot be attributed to lung-brain circulation time alone, but must also involve aerobic or anaerobic reserves in the cerebral tissue.

Oxygen Toxicity

The effects of hypoxia have been discussed at some length. There are other effects of oxygen on the body, some of which are detrimental. These effects are under intensive investigation at this time because of their importance in two critical areas: space flight and submergence. Since naval aerospace medicine actually has interests in both areas, a brief summary of the present status of knowledge may be of value.

Pressures Greater Than One Atmosphere.—Oxygen is toxic to all mammalian biological systems at partial pressures greater than 760mm Hg (one atmosphere). The partial pressure which produces toxic effects varies with the animal but, in general, the greater the partial pressure of oxygen, the shorter is the time before toxic manifestations occur. The toxic effects are produced at a cellular level and are manifested primarily in the central nervous system and the pulmonary system. In humans, central nervous system signs and symptoms occur within minutes at the partial pressure of oxygen which would occur when breathing from an aircraft oxygen system in 100 feet of water. Facial muscle twitching, nausea, dizziness, anxiety, confusion, and incoordination may occur before the onset of grand mal seizures. If the oxygen partial pressure is not decreased after the onset of the convulsion, status epilepticus will occur, with death the outcome (Beckman, 1967).

Usually, consciousness is lost at the onset of the convulsions, and apnea occurs during the event. The convulsion may last for one or two minutes and, in the submerged individual, drowning may occur as a result. Breathing usually resumes spontaneously after the convulsion if the P_{O_2} is decreased or restored to normal, but the patient remains unconscious for several more minutes. A period of 30 to 60 minutes of semiconsciousness then ensues with irrational behavior, great restlessness, and intermittent sleep.

Exposure to moderately increased partial pressures of oxygen, i.e., 760–1,200mm Hg, will produce signs of pulmonary irritation, pulmonary edema, and death, but only after prolonged exposure of from one to four days. The pulmonary effects of oxygen toxicity are, therefore, not of primary significance in an aviation survival situation.

The physiological mechanism of production of "oxygen poisoning" is poorly understood. However, it is known that at O_2 partial pressures above one atmosphere, so much O_2 is carried in solution in the blood that the tissues have a higher O_2 concentration and less O_2 is removed from the hemoglobin. A hypercapnia results which causes cerebral vasodilation with increased blood flow to, and thus increased oxygenation of, the brain. It is believed likely that the oxygen has a direct effect on enzyme systems in the brain, thus causing the convulsion, but the definitive work on this subject in humans remains to be done. Zirkle, Mengel, Harton, and Duffy (1965) showed that in mice, exposure to O_2 at high pressure resulted in lipid peroxidation in the brain, associated with decreased brain acetylcholinesterase activity. The postulated sequence of events leading to the clinical symptoms of O_2 toxicity based upon the work with mice is as follows:

1. Formulation of lipid peroxides in the brain with tocopherol inhibiting the reaction (tocopherol-deficient mice are more severely affected).
2. The formed lipid peroxides then inhibit acetylcholinesterase in the brain.

Treatment consists of bringing the individual up to a normal $P_{O_2(alv)}$ area, while preventing him from drowning. The convulsion itself usually is not immediately fatal. This condition has some importance to naval aviation medicine because aircraft oxygen systems will operate under water in emergencies (see chapter 14, Emergency Escape from Aircraft).

Oxygen Toxicity at Oxygen Pressures of, or Less Than, One Atmosphere.— There are other toxic effects noted in individuals breathing partial pressures of oxygen at, or *less* than, one atmosphere.

These effects are essentially pulmonary and consist of atelectasis, pulmonary edema, and pneumonitis in experimental animals. In man, similar findings have been noted, but again there is a time factor. Comroe, Dripps, Dumke, and Deming (1945) noted that when 100 percent oxygen is breathed at one atmosphere (760mm Hg), substernal distress appears after 12 to 16 hours. However, if the breathing is performed at greater altitudes (or lesser pressures of 100 percent oxygen), no ill effects occur. It is believed that these effects are due to the direct toxic action of the oxygen on the lung and may possibly be due to the effect on alveolar surfactant.

Pulmonary surfactant is thought to stabilize the alveoli and protect them against an inherent tendency for collapse or atelectasis. It is also thought to protect the alveoli from pulmonary edema and hemorrhage and has been found to be missing in cases of "oxygen poisoning." The experimental work on surfactant is in very early stages and must be viewed with caution.

Acceleration Atelectasis.—A final oxygen toxicity effect is atelectasis occurring while breathing 100 percent oxygen under acceleration, although the term "oxygen toxicity" in this context is a complete misnomer. However, acceleration atelectasis is included in this section because it is considered to be an adverse effect of breathing 100 percent oxygen. Field trials conducted by Green and Burgess (1962) demonstrated that repeated acceleration-producing maneuvers in flight cause a marked reduction in vital capacity, along with basilar atelectasis by X-ray when 100 percent oxygen is breathed. They showed that when a 50 percent O_2/50 percent N_2 mixture was breathed, only a minimal reduction of vital capacity and minimal radiological signs of collapse were present after a similar flight.

As noted by Ernsting (1964), the primary factor responsible for the atelectasis is probably the complete cessation of the basilar alveolar ventilation under acceleration. When under acceleration, there is also markedly increased blood flow to the basilar alveoli as opposed to the apical ones, along with a reduction in basilar alveolar volumes due to the weight of the lung under acceleration compressing the bases against the diaphragm. With these three factors acting in concert and when the alveoli in question contain only O_2, H_2O vapor, and CO_2, oxygen absorption (the main cause of acceleration atelectasis) leads to alveolar collapse and atelectasis can occur very rapidly.

If N_2 is present in the inspired gas, however, the gas absorption and consequent alveolar collapse are greatly slowed. Ernsting states that the time required for complete absorption of gas contained in the lower quarter of the unventilated lung (with normal blood flow distribution) is increased from five minutes on 100 percent O_2 to about 25 minutes on 50 percent O_2/50 percent N_2. In addition, some recent work gives evidence that N_2 in the lung acts as a "spring" by preventing alveolar collapse when all the O_2 is absorbed.

Pulmonary atelectasis during flight has several detrimental effects, among them, distracting or perhaps even incapacitating cough and chest pain, and arterial hypoxia due to the shunt of venous blood through the nonaerated alveoli.

Ernsting and others have suggested the use of N_2/O_2 mixtures to prevent atelectasis. The experience of the Navy to date has been that atelectasis, although it occurs, has not been a problem because prolonged accelerations are required to produce it, and these would occur only in dogfighting for several minutes. In earlier aircraft using the diluter-demand O_2 regulator, acceleration atelectasis was not a problem at low altitudes because the O_2 was diluted with air. New aircraft using liquid O_2 converters and miniregs do not dilute the O_2 with air, even at low altitude, so that conceivably acceleration atelectasis would be a hazard on missions requiring accelerations. The Flight Surgeon should remain aware that coughing, substernal pain, and decreased altitude tolerance can be the result of this mechanism.

Acceleration atelectasis usually resolves itself in a few days or less with no treatment.

Operational Problems with Hypoxia

Within some sections of the naval aeronautical organization, behavior of O_2, CO_2, N_2, and H_2O vapor seems unimportant. The ASW helicopter in normal missions ordinarily does not exceed an altitude of 1,000 feet. Marine Corps helicopters may have even lower ceilings for normal missions. The ASW fixed-wing aircraft in many cases will not exceed the altitude limits noted for ASW helicopters during an entire deployment. Many Navy transport aircraft have pressurized cabins. New tactical doctrines for employment of transonic attack/fighter aircraft or even supersonic fighter aircraft may have them coming in to a sea level target at a maximum altitude no greater than that of the ASW helicopter.

Why then is the Naval Flight Surgeon required to study hypo- and hyperbaric oxygen physiology when many of these aircraft do not even possess O_2 systems? The answer is that anything which affects respiration is of interest to the Flight Surgeon. Smoking cigarettes affects oxygen physiology, especially at night. Carbon monoxide from any other source, such as defective exhaust manifolds or cabin heater, affects oxygen physiology. The aircraft which is normally flown at altitudes of less than 1,000 feet must be flown in excess of 14,000 feet when crossing the Rockies and usually is flown at altitudes of 10,000 feet or above in going from north to south along the crowded air corridors of the eastern seaboard of the United States. Since maintenance of an adequate P_{O_2} (as well as the NATOPS rule) requires the use of O_2 above 10,000 feet, even ASW aircrews can be afflicted by hypoxic episodes.

Navy aircraft fly over water, and the physiology of hyperbaric O_2 and N_2 must be at every pilot's fingertips lest he suffer injury or even death during rescue from submerged aircraft.

Finally, aircraft with high-altitude, high-speed capabilities will have those capabilities used in the event of conflict with any major power. It is the duty of the Flight Surgeon to insure that capabilities of aviators are not restricting factors on utilization of the maximum performance of the aircraft when such conflict appears.

Such a conflict might begin on very short notice, and both the aviator and the Flight Surgeon must be prepared to meet the challenge. To the line commander, readiness might include instant availability of fitted and operational full pressure suits or antiexposure suits. For the Flight Surgeon, readiness would include having aircrew training in the use of such equipment as well as in the physiological bases of operations in the required environments up to date at all times.

Any time a pressure cabin is being utilized, there is the danger of rapid or explosive decompression due to equipment failure, structural failure, or human error. In this situation the transition from the quiet and ease of high-speed flight to a brutal environment is quite rapid. If equipment fails or if training has been inadequate, the man, the aircraft, or both may be lost.

The final, and one of the most important and compelling reasons for the Flight Surgeon's study of gas physiology is to assure mission completion. Any recently designed aerospace craft has redundancies in its important systems to insure reliability. Many of the newer jet fighter and attack aircraft have multiple engines, multiple navigational systems, multiple communications systems, multiple weapons release modalities, and various emergency units in addition to the redundancies.

In an aircraft which is designed and constructed to be capable of sustained powered flight above 50,000 feet at supersonic speed, two oxygen supply redundancies are presently being used. One is the pressure cabin (which was described earlier) and the other is the liquid O_2 converter, regulator, and mask. In peacetime operations, the two systems used together provide a pleasant working environment, excellent safety, and minimal work requirements in mission completion.

In a combat situation, however, any hole in the pressure cabin from a missile near-miss, hostile fire, or blown canopy would necessitate descent to and maintenance at or below 44,000 feet. Any defect in the cabin pressurization system would have the same effect. To counteract these threats and to provide a much higher probability of mission success, the Navy has equipped fighter and attack aircraft for full pressure suit use. If the cabin pressure were to fail for any reason, including hostile fire, the suit controller would maintain the aviator at 35,000 feet, breathing 100 percent O_2, allowing him to complete his mission without interruption.

Information about the utilization of the full pressure suit is to be found in the NATOPS Manual for each aircraft in which it can be utilized.

Hypoxia Prevention

All jet fighter/attack/reconnaissance aircraft require use of oxygen masks during the entire flight. To insure that the oxygen system performs its function, pilot training must reflect the following preventive measures:

Oxygen System Maintenance and Inspection.—Pilots must understand the functioning of oxygen equipment. Under no circumstances should a leaking mask be used.

Scrupulous cleanliness of the oxygen mask must be maintained. It is recommended that masks be cleaned every week if in constant use and once a month, even if not used at all.

A thorough preflight inspection should be made of the oxygen mask prior to each flight. This should include at least a check of the general condition of the mask and its connection, and a check to see that both inhalation and exhalation valves are properly seated and hose connections are adequate. The pilot should be certain that he can inhale and exhale normally through the mask prior to manning his aircraft.

Finally, a thorough preflight inspection should be made of the aircraft oxygen system.

Emergency Procedures.—Standard emergency procedures to be followed in the event of suspected hypoxia are:

Immediately select 100 percent oxygen if using a diluter-demand regulator.

Immediately descend to 10,000 feet or below. (This is the altitude at which to check for possible malfunctioning of the equipment. Such checks should not be performed at the altitude at which hypoxia is first suspected.)

Hypoxia Detection by Others.—Marine Corps and Navy tower operators as well as other aviators should report to the Flight Surgeon *as an emergency* any incidents of incoherent or seemingly intoxicated speech which they detect in air-to-ground or air-to-air communications. This may be the only evidence of hypoxia and occasionally some assistance can be given the aviator by radio.

Aviators should be encouraged to report every instance of severe headache or period of extended lethargy following a high-altitude flight. Such incidents call attention to possible attack of hypoxia and possible equipment malfunctions of which the pilot may be completely unaware. The necessity of reporting all cases of known or suspected hypoxia, should be stressed to all pilots, even though recovery is apparently immediate and complete.

Remember, there are no chemical tests, physical findings, or any other findings to confirm a case of hypoxia once the flight is over and the aviator is available for examination on the ground.

Investigation of Suspected Hypoxic Incidents.—The Flight Surgeon should meet the aviator when he lands his aircraft after a suspected hypoxic incident. If the Flight Surgeon has had the opportunity of getting to know the aviators in his unit, he may well note evidence of some residual confusion following a hypoxic episode simply by engaging the pilot in conversation about mutually familiar subjects. A mental status examination and neurological examination should be performed as soon as possible, and blood pressure and pulse rate should be recorded. Then a thorough history should be taken of the suspected incident, obtaining as many particulars as possible. These should include:

1. Symptoms, signs, and reactions during the incident—effects of emergency measures.
2. Cabin altitude and duration.
3. Type of mask, ownership, size, and fit.
4. Type of regulator and regulator control settings.
5. Duration of flight.
6. Possible predisposing factors—smoking, hangover, scuba diving, anxiety, CO exposure.

The classic medical procedure of first obtaining a chief complaint, then a history of present illness, past illness, and review of symptoms, followed by the physical examination, must be reversed in these investigations. If the history is taken before the neurological and mental status examinations are performed, the occasional residual deficit will be missed.

Concurrent with the physical examination and history, expert maintenance personnel should be checking the aircraft O_2 system for normal function, O_2 quantity remaining, regulator control settings, and evidences of leaks, disconnections, or faulty gauges.

After allowing the aviator some rest, the Flight Surgeon should have him don the O_2 mask and helmet and should proceed to check the fit of the mask on the aviator's face. The mask should then be examined thoroughly for function by the Flight Surgeon: (1) exhalation, (2) inhalation, and (3) leaks.

If the entire procedure reveals evidence of a hypoxic incident and/or its cause, responsible line officers should be notified immediately so as to preclude similar incidents, possibly even in aircraft then airborne.

Causes of Hypoxic Incidents.—Mask removal for convenience is one of the most common causes of hypoxic incidents. Aviators sometimes remove their oxygen masks to adjust a visor or to scratch their beards. Because of the insidious nature of hypoxia, such actions are extremely hazardous. When replacing the mask after only a few moments, the aviator may be so uncoordinated that he cannot perform the simple motions required and can never get the mask replaced. He may have such impairment of judgment that he thinks the mask is properly reattached, when in fact it is not; or he may think it is no longer necessary to reattach the mask at all.

Should brief removal of the mask from the face be necessary at high altitude, the following procedure should be followed:

1. Take three or four deep breaths of 100 percent oxygen.
2. Hold breath and remove mask from face.
3. As soon as praticable, replace mask and take three or four deep breaths of 100 percent oxygen.

Inhalation valve problems in the A-13A oxygen mask often cause hypoxic incidents. The *in*let valves of this mask are very sensitive to dirt. A small particle between the flapper valve and its seat (see malfunction D of figure 11-10) which prevents complete closure will result in a marked increase in the *ex*halation effort required. The aviator usually does not know enough about his mask to realize that the *in*halation valve is almost invariably the villain in *ex*halation problems. When the inlet valve is held open by a particle of dirt, the exhalation pressure exits through it and is then exerted on the rear of the exhalation valve, exactly balancing the pressure on the front of the exhalation valve and preventing it from moving, regardless of *ex*halation pressure exerted by the aviator (see malfunction B of figure 11-10). When faced with such marked exhalation difficulty in flight, the aviator sometimes panics and removes his mask in the belief that he may have exhausted his oxygen supply. Indications that he has *not* are the movements of the oxygen flow meter in some oxygen systems, the oxygen quantity gauge reading in all systems, and the fact that inhalation is still perfectly normal. Logic rarely triumphs in the face of panic, however, and training is the only way to preclude such panic.

If exhalation becomes difficult or impossible in flight, the aviator should keep his mask in place. When exhalation is desired, he should vent the expired air past his chin and out of the mask. With appropriate movement of the mandible and a little training, this is easily accomplished.

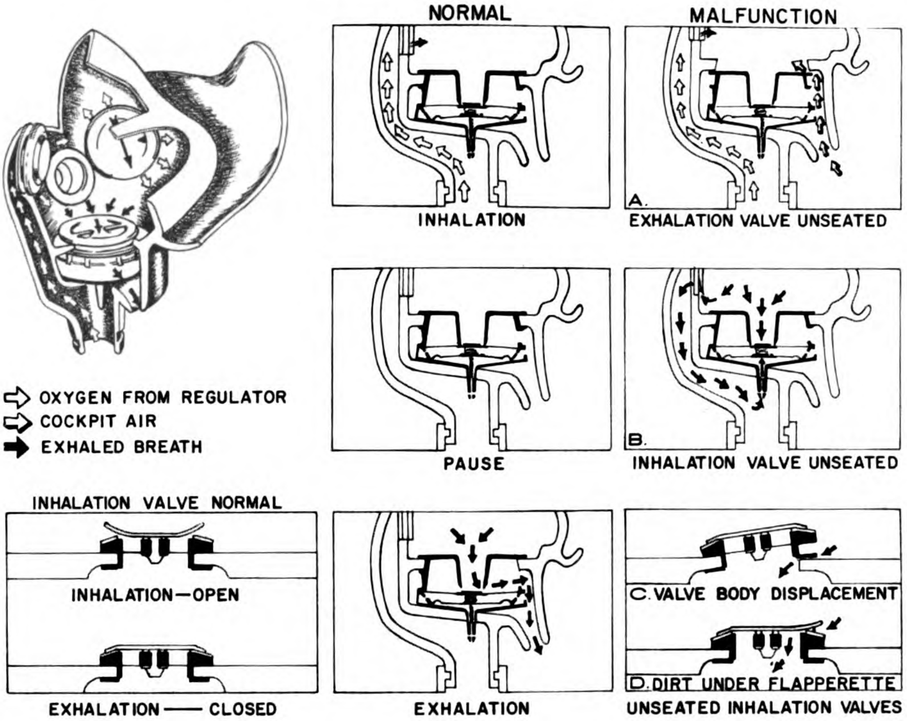

FIGURE 11–10. *The A-13A oxygen mask, normal, and with malfunction.*

Exhalation valve problems in the A-13A oxygen mask also may be the cause of hypoxic incidents. Functional failure of an exhalation valve is extremely rare. As has been pointed out, all exhalation difficulty is due to malfunctions of the *inhalation* valve. However, the exhalation valve *can* be the cause of a dangerous inflight situation. The exhalation valve can be completely unseated, and most oxygen equipment technicians would not detect it, nor would most Flight Surgeons or aviators. The interior of the mask appears to be just as normal with the exhalation valve completely unseated as with it properly seated. Figure 11–11 is the normal mask. Figure 11–12 shows the completely unseated exhalation valve. The mask in figure 11–12 still will pass tests for function in such a way as to appear satisfactory to most users. The hazard lies in the fact that when it is in the unseated position, inhalation can permit cockpit air to enter the mask around the exhalation valve, thus decreasing the oxygen concentration in the mask to some extent, as noted in malfunction A of figure 11–10. Since 100 percent oxygen would still enter the mask through the inhalation valves, the aviator would not become hypoxic at medium flight levels, nor would he detect any difficulty in exhalation or inhalation. However, when attempting to reach the limits of altitude for which the mask is designed, he would become hypoxic at considerably lower altitudes. This effect is made worse by any action such as head turning or any maneuvers producing accelerations, because such actions cause some distortion of the mask and effectively increase the leak aperture and thus amount of cockpit air inhaled. Again, the only solution to this problem is prevention. The aviator must be trained to recognize an unseated exhalation valve instantly during preflight inspection, and trained to *look* for it before the flight.

Another frequent reason for removing the oxygen mask at altitude is the fear of "contaminated oxygen." Cases of true contamination of the oxygen supply as a cause of hypoxia, illness, unconsciousness, or any other adverse physiological effect are so rare in Navy and Marine Corps aviation as to be almost unheard of. Yet the pilot who sniffs an unusual odor in flight typically feels his oxygen supply is contaminated and removes the only source of oxygen he has, usually with dire consequences. Bartlett (1961) demonstrated that, in certain sensitive aviators, the nose can detect odors of substances in concentrations undetectable by any other means short of spectrometry. Since the nose is so sensitive, it can sometimes detect "trace" contaminants in an aviator's breathing oxygen that are impossible to exclude completely in the manufacturing process but which are absolutely harmless.

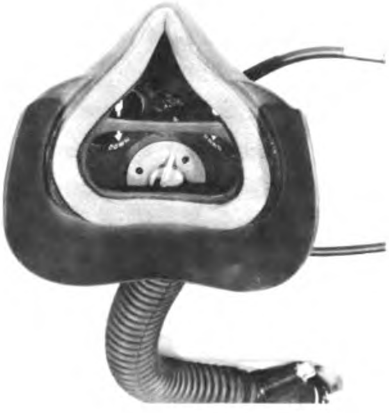

FIGURE 11–11.

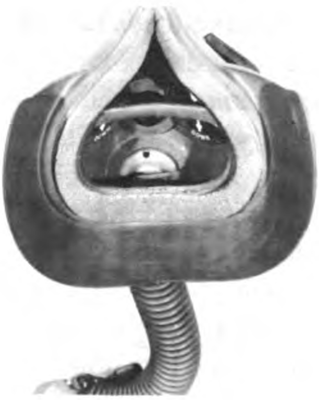

FIGURE 11–12.

Which mask is abnormal, and why?

The only answer to the "contaminated oxygen" problem lies in training, both of the Flight Surgeon and of the aviator. Salient facts are these:

1. For all practical purposes, contaminated oxygen does not exist in the sense that it can cause any harmful effect to the aviator.
2. The major harmful effect that occurs from such incidents is hypoxia resulting from removal of the mask.
3. The procedure to follow if such a situation arises is to select 100 percent oxygen if using a diluter-demand regulator and to trust the equipment if using a full pressure suit or miniature regulator. If odor continues, or nausea due to the odor ensues, descend to 10,000 feet or below, possibly activating bailout O_2 bottle en route, and then remove the mask if necessary.

Accidental mask detachment can result in hypoxia. Marine Corps and Navy attack and fighter pilots operate in a very dynamic environment, pulling high G maneuvers, moving about in cramped cockpits in order to get comfortable, or maintaining a lookout for other aircraft, missiles, birds, etc. Occasionally a quick-disconnect fitting will disconnect accidentally, causing an emergency until it can be reconnected. If the cabin altitude is fairly low, such disconnection may not become apparent for some time and the crewmember will suffer gradual onset of the early symptoms of hypoxia. In cases where the cabin is at ambient pressure, such symptoms can manifest themselves in as short a time as 12 seconds (above 45,000 feet). To give the aircrewmember a chance to reconnect the mask before losing consciousness, the quick-disconnect of the A–13A mask has been fitted with a valve, actuated as soon as the mask is disconnected, which causes a marked increase in *inspiratory* effort as a warning. If a miniature regulator is being utilized, the warning is conveyed by the fact that no breathing is possible at all. In the full pressure suit, warning of a possible disconnection is conveyed by a complete inability to breathe, but only if the face seal is tightly closed and if the visor seal is tight. Oxygen-want warning systems have been designed and evaluated but do not, at present, offer sufficiently great advantages to warrant their universal adoption.

Inexperienced personnel collapse more frequently at intermediate altitudes when hypoxic than do experienced personnel (Boothby et al., 1954). An individual who is apprehensive concerning a high-altitude flight may, under a mild attack of hypoxia, hyperventilate to such an extent as to produce a hypocapnic reaction which can lead to total collapse or to a serious lessening in motor coordination. In one instance, a passenger on a first jet ride experienced difficulty with his oxygen equipment, finally turning off the oxygen supply in his struggle to relieve breathing difficulty. The passenger became unconscious and died in spite of the pilot's rapid descent and the immediate availability of medical aid upon landing.

In summary:

1. Almost all exhalation problems are caused by the inhalation valves.
2. Inhalation difficulty is always due to a disconnected hose or exhausted O_2 supply.
3. When hypoxic incidents occur, look for unseated exhalation valves.
4. Contaminated oxygen as a cause of hypoxia has never been found in the Navy.
5. Training and inspection are the keys to avoidance of hypoxia in aviation.
6. The Flight Surgeon will rarely obtain any positive laboratory or physical findings in a case of suspected hypoxia. All such cases should be investigated as thoroughly as humanly possible *anyway*.

Provision of Oxygen

The physiological requirements for the provision of oxygen can be generally stated as follows:

1. The concentration of oxygen delivered to the aviator must be adequate to maintain arterial oxygen saturation within design limits.
2. Gas flow capability must meet the full range of respiratory flow requirements.
3. The resistance to flow should be minimal to provide a system which is as "natural" as possible.

All oxygen systems consist of (1) a supply of oxygen, (2) the necessary tubing or hoses to distribute the gas, (3) a means of controlling or metering the flow of the gas (regulator), and (4) a means of directing the oxygen to the respiratory system without inadvertent leakage where the man meets the system.

Oxygen Supply Systems.—There are two types of oxygen supply systems currently in use in the Fleet: bottles (both low pressure and high pressure) and liquid oxygen (LOX) converters. The oxygen bottles have almost disappeared from use in first-line Fleet aircraft. Some obsolescent training and transport aircraft still use one or the other of these types, and bailout bottles or walkaround bottles are another exception.

The LOX converter is universally utilized in operational jet aircraft because of its advantage in weight and storage space over a bottle system. For example, a five-liter LOX converter weighs 28.6 pounds as opposed to 90 pounds for a high pressure oxygen bottle of the same capacity and requires only 0.7 cubic feet of space as opposed to 3.6 cubic feet for the high pressure bottle system.

The range of an aircraft with the capability of flying at altitudes requiring oxygen breathing by the crew is a function of the amount of consumables aboard. These include aircraft fuel, lubricating oil, drinking water, food, and oxygen supply. The oxygen supply is, in fact, the limiting factor on aircraft range since air-to-air refueling has made it theoretically possible for combat jet aircraft to remain airborne until their engines need overhauling.

The NATOPS Manual for each aircraft contains a table of O_2 duration which is used for mission planning. Table 11–14, for example, is the oxygen duration chart for the F–4B aircraft. The chart is based on utilization of 100 percent oxygen at all altitudes and takes into account evaporation losses which are to be expected from the LOX system. It presumes a value for ventilation rate somewhat in excess of that normally encountered but takes into account the increased ventilation rate to be expected due to apprehension or other factors in the operational situation.

TABLE 11–14. *Oxygen Duration, F–4B Aircraft*

Oxygen duration-hours

	Cabin pressure altitude feet	Gauge quantity-liters						
		10	8	6	4	2	1	Below 1
Duration time is halved when two crew members are using oxygen	40,000 and Above	60.6	48.5	36.4	24.2	12.0	6.0	Emergency—Descend to altitude not requiring oxygen
	35,000	37.0	29.6	22.2	14.8	7.4	3.6	
	30,000	27.2	21.8	16.4	10.8	5.4	2.8	
	25,000	20.4	16.4	12.4	8.2	4.0	2.0	
	20,000	16.0	12.8	9.6	6.4	3.2	1.6	
	15,000	12.8	10.2	7.6	5.2	2.6	1.2	
	10,000	10.0	8.0	6.0	4.0	2.0	1.0	
	5,000	8.4	6.6	5.0	3.2	1.6	0.8	
	SEA LEVEL	7.0	5.6	4.2	2.8	1.4	0.6	

(NAVAIR 01–245FDB–1, 1 Oct 1965.)

Liquid oxygen containers are spherical "Thermos" bottles with double-walled vacuum insulation. Some of the valves used in the liquid oxygen system are manually operated; others are automatic. The valving controlling the system pressure, for example, is automatically actuated to maintain a system pressure of 70 psi. The filler valves, on the other hand, are operated manually to allow an initial filling at atmospheric pressure. This is followed by a system pressure buildup which achieves the required 70 psi. An automatic pressure-relief valve on the converter positively prevents excessive pressure buildup, thus precluding the danger of an explosion.

If system pressure becomes excessive, the relief valve has a cracking pressure of 100 psi and a full flow pressure of 120 psi. Approximately one liter of liquid oxygen will be lost through this valve and overboard during a 24-hour period when the system is not in use.

Tubing for liquid oxygen systems is of aluminum, a lighter material than the copper tubing used for gaseous oxygen. Because the liquid oxygen operates under relatively low pressure, the system tubing need not be as strong as that used in gaseous oxygen systems. In the LOX system, the tubing and its component valves make and maintain system pressure. When liquid oxygen is converted to gaseous oxygen in the tubing of the converter, it expands. When expansion creates a system pressure of 70 psi, a valve shuts off excessive oxygen from the system. Excessive gaseous oxygen is carried to an overboard vent through the system's tubing.

Safety Precautions in Handling LOX.—Oxygen does not burn, but it supports combustion. When the percentage composition of oxygen in an atmosphere rises, the burning rate of most materials also rises. When 100 percent oxygen is the only component of the atmosphere, traditional safety measures are no longer adequate to assure safety. The kindling points for many materials are revised drastically downward.

Specifically, *any* hydrocarbon in the form of oil or grease may combine with 100 percent oxygen *explosively* at room temperature. Any trace of oil or grease anywhere in the oxygen breathing system may cause a sudden fire, perhaps leading to destruction of the aircraft when LOX or compressed 100 percent oxygen comes in contact with it.

Since maintenance crews and aviators live and work with LOX and 100 percent oxygen daily, there is a tendency gradually to relax safety precautions. Familiarity with the substance does not alter its properties one iota. Recently, an experienced aviator returned from a flight with most of his oxygen mask melted and severe burns over the side of his face, caused by his lighting a match "to get rid of some raveled strap ends." (See figure 11–13.) He was known to be a heavy smoker and had cigarettes and matches in his flight suit. The number of fires resulting from smoking while wearing the oxygen mask will never be known, since most such incidents leave no trace at the scene of the crash. Smoking while oxygen equipment is in use is strictly forbidden by the NATOPS Manual, OPNAVINST 3710.7 series.

As another example, during an experimental trial in a 100 percent oxygen atmosphere of a new life support system in a low pressure chamber, one of the subjects attempted to remove a light bulb from the electrical socket using a bath towel to shield his hands from the hot bulb. The normal

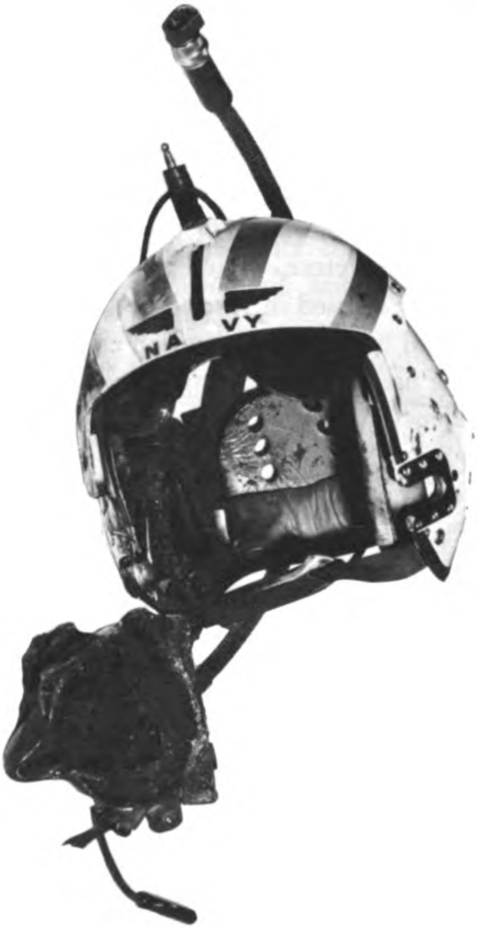

FIGURE 11–13.

operating temperature of the light bulb was sufficient to ignite the towel in the 100 percent oxygen atmosphere. The resulting fire injured several persons.

Even worse, liquid oxygen, when combined with other substances such as liquid or powdered soaps, sawdust, cloth, and many others, forms a gel within a very short time. If the mixture is then subjected to an impact, such as a hammer blow, it will explode with a force roughly equivalent to a similar weight of TNT. In one example of this most unexpected and little-known quality of LOX, the technician for a commercial firm supplying LOX to community hospitals had his leg blown off without warning or obvious cause. Investigation revealed that the event occurred on a concrete-surfaced alley, down the center of which many vehicles over the years had dripped oil. When some of the LOX being transferred dripped onto the pavement no notice was taken of it. However, when the technician stepped on the site of the spillage, the gel of old crankcase drippings and LOX had formed and the impact of his footstep was enough to cause the explosion.

It must be reemphasized that cloth impregnated with LOX may give an identical reaction. If, in addition, the clothing of a maintenance man has been soiled with oil, hydraulic fluid, or grease, and LOX *is* spilled on it, tragedy may result. Therefore, maintenance personnel handling LOX should be immaculately dressed in special clothing; all possible leakage or spillage of LOX should be prevented; and all clothing should be laundered after each occasion of use. The following procedures should be followed to avoid injury or accident:

Use only that equipment which is especially provided for use with LOX. The extreme cold of LOX will instantly produce painful burns if it is held in contact with the skin. Protective equipment, consisting of a suitable face shield, apron, and gloves must be worn when handling LOX. Avoid touching bare metal lines containing LOX. Bare skin will instantly freeze to the —297° F metal.

LOX must be added slowly to a completely empty system so that temperatures will not drop too rapidly. Equipment may be damaged by thermal shock or excess pressure if the LOX is forced in too rapidly.

Oxygen Quantity Gauges.—The gaseous oxygen flowing from the liquid oxygen converter moves to the pilot under low pressure. The pressure of the gaseous oxygen is maintained by means of the liquid oxygen conversion system, which maintains the delivery pressure and volume by the controlled evaporation of liquid oxygen. Information on the pressure of the gaseous oxygen in the system is provided the pilot by a panel-mounted pressure gauge similar to the pressure gauge for nonliquid oxygen systems. The only difference is the calibration of the gauge. For the *gaseous* oxygen system, the gauge is calibrated from 0 to 1,800 psi. For the low pressure *liquid oxygen* system, the gauge is calibrated to read from 0 to 150 psi. Since the pressure of gaseous oxygen in the liquid oxygen system is normally only around 70 psi, the 0-to-150 gauge permits easier reading of the system pressure.

An electrical capacitance gauging system gives the pilot an accurate means of determining the amount of liquid oxygen remaining in the converter at any time.

Oxygen Regulators.—The oxygen regulator serves several purposes: it meters the amount of oxygen supplied to the user; it regulates the pressure of inspired gas reaching the mask, keeping it within certain design limits; and it automatically changes the pressure at which inspired gas is supplied to the mask in accordance with changes in barometric pressure. In some systems, it automatically increases the Po_2 in the inspired gas as required by decreasing barometric pressure.

Types of Oxygen Regulators.—Currently, there are four types of oxygen regulators in use in Navy aircraft: automatic continuous flow, positive pressure diluter-demand, diluter-demand, and miniature 100 percent oxygen demand or "minireg."

Many older aircraft of the transport, training, combat-readiness training, and even advanced jet trainers use the positive pressure diluter-demand regulator. Only the A–13A mask can be used with this type of regulator. With the diluter-demand regulator, without positive pressure, the A–14 mask may be used.

The A–14 mask and diluter-demand regulator are rarities today, and the automatic continuous flow regulator is also obsolete. For purposes of this manual, therefore, discussion of oxygen regulators will be limited to a short presentation of principles of diluter-demand regulator design, and a much more complete presentation of the minireg, which is in use in almost all of the Navy's jet attack/fighter aircraft today. Details of other oxygen regulators and systems can be found in *Safety and Survival Equipment for Naval Aviation,* NAVAER 00–80T–52 and/or in NATOPS and maintenance manuals for the specific aircraft.

Design Principles of Diluter-Demand Positive Pressure Oxygen Regulators.—It is possible, up to 33,000 feet, to maintain a $Po_{2(alv)}$ identical to that at sea level, breathing air (or about 100mm Hg), by adding oxygen to the ambient air in sufficient quantity to overcome the decrease in $P_{O_2(alv)}$ due to decreasing barometric pressure. At 33,000 feet, breathing 100 percent O_2, the barometric pressure is equal to $P_{O_2(alv)}$ at sea level, and therefore $P_{O_2(alv)}$ begins to decrease as ascent is continued above 33,000 feet. At 39,000 feet, breathing 100 percent O_2, barometric pressure is equal to $P_{O_2(alv)}$ at 10,000 feet, breathing air, so that this is the ceiling for nonpressurized aircraft using only a diluter-demand regulator.

Actually the diluter-demand regulator begins furnishing 100 percent oxygen at 27,000 feet rather than 33,000, as noted previously. In addition, 100 percent oxygen can be selected at sea level, in which case the diluter feature is removed from the system and ten percent oxygen is breathed continuously.

Early oxygen systems delivered a constant flow of oxygen to the mask of the user. This was extremely wasteful and the demand system, which delivers oxygen only during the inspiratory phase, was developed. The regulator senses the slight decrease in pressure as inhalation begins and this initiates delivery of oxygen to the mask.

The diluter-demand principle has several advantages:

1. It saves oxygen during climbout and descent and thus prolongs oxygen duration.
2. It prevents to a considerable degree the development of oxygen otitis media.
3. It prevents the effects of "oxygen toxicity" (i.e., acceleration atelectasis) when utilized at lower altitudes during acceleration-producing maneuvers.
4. When utilized at altitudes below 27,000 feet, it prevents the severe drying effects of 100 percent oxygen on the respiratory tract.
5. It has all the features of 100 percent oxygen demand regulators if selected.
6. It is inexpensive.

Disadvantages of this regulator are that:

1. It has a considerable size and weight disadvantage compared to the minireg, at least at present stage of development.
2. Since the advent of LOX converters, oxygen duration is of less importance than formerly.
3. If the diluter feature is selected, denitrogenation is not accomplished as rapidly during climbout.
4. It cannot be used for underwater breathing if the diluter feature is selected at time of crash.

The 100 Percent Oxygen Demand Pressure Breathing Regulator (Minireg).—The minireg is also a demand regulator but does not have the diluter feature. One hundred percent oxygen is automatically delivered to the mask under a slight positive pressure (called "safety pressure" in diluter-demand regulators) of 1.5 inches of water (2.8mm Hg) at all flight levels. The slight positive pressure is beneficial in two ways: it decreases inspiratory effort and thus lessens fatigue, and it precludes inboard leakage of cabin air or toxic gases. The minireg is normally mounted to the A–13A oxygen mask. The short mouth-to-regulator distance thus produced reduces the dead space and thus the resistance to flow, thereby affording rapid response to inspiratory pressures.

The increased pressure of oxygen needed at pressure breathing altitudes is accomplished by an aneroid. At low altitudes, a small induced leak of oxygen is vented through the regulator. As altitude increases, the aneroid expands, gradually closing the vent exhaust port. The restricted flow applies a pressure to the diaphragm through increasing the mask pressure and providing more oxygen. If for any reason the pressure in the mask should get too high, a pressure relief valve opens. It stays open until pressure is reduced.

The regulator operates on 100 percent oxygen at pressures from 50 to 150 psi. With this inlet supply pressure, the regulator is capable of automatically maintaining a positive pressure not exceeding two inches of water, for ambient flows up to 100 liters per minute, when measured at altitudes from 10,000 feet to 35,000 feet. The automatic pressure breathing element permits the regulator to deliver positive pressure in accordance with table 11–15.

TABLE 11–15. *Positive Pressure Loading at 10 LPM Ambient Flow*

Positive pressure (inches of water)		*Altitude in feet*
Minimum	*Maximum*	
	3.5	35,000
	5.7	37,000
	8.0	39,000
2.0	9.4	40,200
4.0	10.2	41,000
6.0	10.8	41,500
8.0	12.0	42,500
10.0	12.5	43,000
	18.0	(1)

[1] 50,000 feet and above. 1 inch water=1.87mm Hg.

(NAVAER 00-80 T-52)

Advantages of the minireg are that:

1. Denitrogenation automatically begins as soon as mask is donned.
2. All takeoffs and landings automatically occur while breathing 100 percent oxygen. (For a discussion of this very important feature, see the section on emergency escape from aircraft underwater.)
3. On ejection or bailout, the regulator goes with the pilot, whereas the older, larger regulators stayed with the aircraft. The pilot thus is automatically supplied with a regulated flow of oxygen from the bailout bottle during the whole process of ejection, rather than an unregulated high pressure continuous flow. (Again, see the section on emergency escape from aircraft underwater.)
4. An excellent oxygen-want warning is inherent in the design of the minireg. If oxygen is not being supplied, one cannot breathe. In the diluter-demand regulator at altitudes lower than 27,000 feet, the diluter feature would supply air alone if the oxygen supply ran out, thus giving no warning of hypoxia since breathing appeared normal. In the event the oxygen supply level of the minireg approaches the usable minimum, breathing resistance gradually increases. If this inhalation resistance went unnoticed, there would eventually be no flow when the oxygen pressure got below 15 psi. In such an event, the pilot could simply activate the emergency oxygen bottle and descend safely to below 10,000 feet. This system represents a great advance in flight safety over earlier designs and is typical of the excellent engineering solutions to physiological and psychological problems in aviation.

Disadvantages of the minireg are that it:

1. Requires use of 100 percent oxygen at altitudes where breathing air would be physiologically sound, thus causing oxygen waste.
2. Increases incidence of oxygen otitis media.
3. Increases incidence of acceleration atelectasis.
4. Causes severe drying of respiratory epithelium with some degree of altered physiology resulting.
5. Is expensive.

For complete information concerning oxygen regulators installed in all Navy and Marine Corps aircraft, reference should be made to NAVAER 00–80T–52, *Safety and Survival Equipment for Naval Aviation;* the *Parachute Rigger's Manual;* and to the maintenance manual(s) for aircraft operated by the squadron.

Oxygen Masks.—There are several means presently used in the Navy to supply oxygen to the respiratory apparatus: the A-13A pressure breathing mask, the A-14 mask, and the MK IV full pressure suit helmet.

The A-13A pressure breathing mask is composed of several parts:

1. Body.
2. Inhalation valves (with covers).
3. Exhalation valve.
4. Oxygen supply hose, with or without minireg.
5. Nosepiece clamp.
6. Face seal.
7. Microphone.
8. MC-3A connector (where minireg not installed).
9. Suspension system.

The body of the mask is supplied in three sizes: small, medium, and large. It is a standing joke in some naval air activities that the three sizes are, instead, "too small," "too large," and "not in stock." There is a great deal of truth in this, since at least 90 percent of all Navy and Marine Corps aviators use the medium size. The small and large sizes, therefore, are almost universally available, while the heavy usage rate of the medium size results in occasional temporary supply shortages.

It is imperative that only approved and properly fitted oxygen masks be worn by flight personnel. While a Flight Surgeon should know how to fit the mask, the Aviation Physiologist, the parachute rigger, and the survival equipment technician are *better* qualified and in most cases have a great deal more time available to perform such duties. In testing the "fit" of a mask, the individual should mount it on his helmet, and then expire forcefully with one of the inhalation valves removed. If the pressure escapes around the face at moderate exhalation pressures, either the suspension is not tight enough or another mask size is required. At altitude, of course, a much better seal will be available as the facelet seal expands, but this procedure is only used to check the mask fit, not pressure seal.

If the minireg is not mounted, the inhalation valve should then be replaced and forceful inhalation attempted with the supply hose kinked above the quick disconnect. If air leaks in around the sides of the mask, a different size should be selected and tested. If a small size is selected, one must make certain that the hollows of the cheek do not permit inboard leakage during inhalation. If the large size is selected, one must make certain that the upper border of the mask body does not seriously impair vision. Almost no one wears a large size mask, but a few, usually young, aviators wear a small size.

Inhalation valves if unseated or if dirty cause exhalation problems, ***not*** inhalation problems. Exhalation valves if unseated may be an unsuspected cause of hypoxia. (See earlier section on operational problems with hypoxia.)

The oxygen supply hose is often indicated as the cause of oxygen leaks but is rarely the culprit. It should be examined frequently, however, for leaks or deterioration.

The nosepiece clamp should be tightly fastened to hold the supply hose to the mask nosepiece without leaks. If loose, the hose should be pulled to determine whether the "tongue" of the supply hose is still fitted inside the "groove" of the nosepiece. Occasionally, a mask will be found with the clamp tightly fastened but around the hose itself and not around the supply hose-nosepiece junction. This can be a fatal configuration if undetected. This does not apply to the mask-mounted minireg, since it has a different supply hose and is itself fitted *inside* the nosepiece of the A-13A. The clamp fastening the nosepiece to the minireg should be checked for tightness and point of application.

The face seal is a redundancy on the oxygen mask, but an important and lifesaving feature. It consists of a strip of closed-cell plastic sponge cemented around the entire oronasal opening of the mask body. As the barometric pressure decreases, the gases trapped inside the plastic cells expand, creating a tight but not uncomfortable seal, automatically deployed and more efficient the greater the altitude. If, however, the cement deteriorates and portions of the seal become unglued, a potentially hazardous situation is created. No aviator should be allowed to fly with a mask in such condition.

The microphone is especially designed for use in the mask and rarely causes any problem.

The MC-3A connector is used on the distal end of the supply hose when the minireg is not fitted to the mask and a console-mounted regulator is utilized. It has a connector for bailout oxygen supply, connector for aircraft oxygen supply, a webbing strap for connection to the parachute harness, a warning device for unsuspected quick-disconnect detachments (see operational problems with hypoxia), and an attachment to the supply hose. The aviator must be instructed to utilize the webbing strap, placing it ***under*** the shoulder straps and attaching it to the parachute harness. If he does not, bailout or ejection from some of the older aircraft will result in the supply hose being stretched until the quick-disconnect fitting gives way, at which time the disconnector may be slammed into the face by the elastic supply hose.

When the mask-mounted minireg is employed, the nosepiece is nonelastic but flexible, the connector is fastened to the bailout bottle in the seat kit, and thus no strap is needed. The disconnect thus utilized can fit into the same supply sockets for microphone and oxygen as those used for the full pressure suit, thus allowing selection of either mask or full pressure suit for a particular flight.

The oxygen mask suspension system is carefully engineered for many qualities. It is far superior to those in use on the same mask just a few years ago. Rarely are reports seen today of hypoxia suffered by an aviator during the accelerations of a gunnery run or aerobatics flight, due to displacement of the oxygen mask. The suspension is even tested for its ability to resist a windblast of over 500 knots while attached to the helmet, without failure of any part.

The A-14 mask is designed without inhalation valves, so that it cannot provide positive pressure. It is obsolescent although some still are utilized in obsolescent aircraft.

The full pressure suit helmet, or headpiece, as it is officially termed, is essentially the same as an oxygen mask equipped with a miniature regulator. Figure 11-14 shows the components of MK IV FPS helmet.

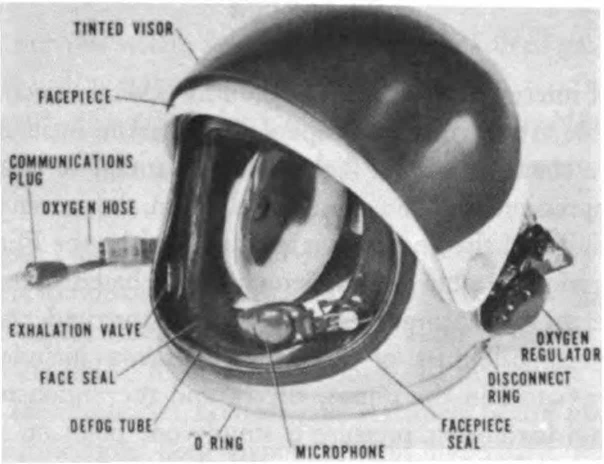

FIGURE 11-14. *Full pressure suit helmet headpiece, MK IV.*

The face compartment has two seals which isolate it from the remainder of the suit (face seal) and from ambient air (visor seal). In normal operation, the helmet is donned, the adjusting knob is turned until the face is pressed into the rubber face seal, and then the visor is lowered and the regulator activated, thus inflating the visor seal. In this way, the face compartment is completely isolated from both ambient air and suit compartment.

The regulator is mounted to the left side of the helmet and is a demand type which provides 100 percent oxygen at 1.87mm Hg pressure in excess of the air pressure in the suit compartment, so that any leakage is from face compartment into suit compartment and not vice versa. Use of the face seal provides a breathing compartment with a small dead space, but leaves the external ears in the suit compartment while the mouth and nose are in the face compartment. This feature occasionally causes some problems when a pressure differential is created by any means between the suit compartment and the breathing compartment (especially since the pressure in the suit may change quite rapidly). The Valsalva maneuver cannot be performed since the nose and mouth are enclosed. Thus, other means of equalizing pressure within the middle ear must be practiced and utilized. Dry oxygen is supplied to the breathing compartment through small holes in the facepiece defog tube and then flows over the visor, serving as a defogging apparatus. On exhalation, a positive pressure differential of 3.7mm Hg must be exerted to cause the exhalation valve to open.

DYSBARISM

Dysbarism is of interest to the Flight Surgeon for several reasons. He must, of course, be able to recognize and cope with dysbarism incidents occurring in low pressure chamber runs in flight. Battle damage to aircraft, leading to rapid decompression, may be a cause of dysbarism, the prevention of which is the responsibility of the line commander, assisted by the Flight Surgeon. Selection of aerospace cabin atmospheres may be based upon the risk of dysbarism inherent to the atmosphere and cabin pressure under consideration. The training responsibilities of the Flight Surgeon include continuous instruction of aviators in the causes, effects, and prevention of dysbarism. Finally, sea level barometric pressure is simply one point on a continuum of pressures ranging from near zero in outer space to the high pressures associated with deep submergence. Man, and especially the naval aviator, may be exposed to all of these, and must either adapt physiologically or be adapted by the design of equipment.

Dysbarism consists of those disturbances in the body exclusive of hypoxia and airsickness, which result from the existence of a pressure differential between the total ambient barometric pressure and the total pressures of dissolved and free gases within the body tissues, fluids, and cavities. In *altitude dysbarism,* the total change in pressure is less than 760mm Hg.

An excellent reference and general review of this subject is H. F. Adler's *Dysbarism,* Review 1-64, USAF School of Aerospace Medicine, Brooks Air Force Base, Texas, February 1964.

Classification of Dysbarism

All symptoms of dysbarism may be considered as being due to the pressure effects of either evolved or trapped gases; however, Adler's classification divides all such symptoms into those due to hypobarism and hyperbarism.

Hypobaric effects result from an excess of gas pressure over ambient gas pressure within body fluids, tissues, or cavities. These effects are subdivided into the evolved gas types and the trapped gas types. Examples of the evolved gas types are:

1. The bends, characterized by joint pains caused by evolved gas bubbles within the joints.
2. The chokes, with cough, chest pain, and dyspnea due to pulmonary intravascular evolved gas, as well as mediastinal emphysema from evolved gas bubbles.
3. Central nervous system symptoms caused by evolved gas pressure on or in the brain, as well as embolic phenomena.
4. Skin disturbances (the itches), due to evolved gas bubbles under the skin and to neurocirculatory phenomena due to bubble pressure on nerves and blood vessels.

Trapped gas types consist of abdominal distention and barodontalgia (covered in the section on dentistry).

Hyperbaric effects result from an excess of ambient gas pressure over that within the body fluids, tissues, and cavities. Examples are barotalgia and barosinusitis (aerosinusitis), both covered elsewhere in this manual.

In this classification, both hypobaric and hyperbaric effects could be suffered on a single flight. The hypobaric effects would be noted on ascent, and the hyperbaric effects would be noted on descent, since the gases in the body fluids, tissues, and cavities are changed during the stay at altitude from those at sea level pressure to the same or different ones at the attained altitude or cabin pressure. Hyperbaric effects would, of course, be noted on any descent below sea level, and hypobaric effects on the ascent from any depth below sea level to any lesser depth. Hypobarism is the major aspect of dysbarism with which this section is concerned.

Etiology

In discussing the etiology of hypobarism, the effects, as noted above, may be divided into those due to evolved gases and those due to trapped gases.

Evolved Gases.—Any tissue in which a dissolved gas is saturated may become supersaturated with a rapid decrease in ambient pressure, thus causing bubble formation. Although N_2 is the gas implicated in almost all cases, O_2 and CO_2 can cause bubbles and thus dysbarism. Also, a nitrogen bubble once formed may become filled with a different gas (such as O_2, H_2O vapor, or CO_2) by diffusion into the bubble. However, for the purposes of the Flight Surgeon, hypobaric effects in the human due to evolved gases may be considered as being due to the effects of the intra- and extravascular formation and expansion of bubbles of N_2 caused by the decrease in ambient pressure incident to ascent to altitude.

The total amount of N_2 normally in solution in the body is 1,000 to 1,500 milliliters, representing saturation of the tissues with this gas at sea level. Almost all of this is in solution in the tissues, with a comparatively small amount in solution in the blood.

Nitrogen is an inert gas and has no metabolic effect. While CO_2 and O_2 are transported in the blood both in chemical combination and in solution, N_2 is transported only in simple solution according to Henry's Law. Nitrogen enters into, and is removed from, the body by the establishment of an equilibrium between N_2 pressures in the alveolar air and the venous blood, and another between the tissues and the arterial blood.

When ambient P_{N_2} is reduced, as during ascent, N_2 transport is directed toward removal from the lungs. The tissue P_{N_2} is higher than that of the arterial blood, so tissue N_2 is lost to the blood, and thus to alveolar air, and thus eventually to outside. However, only a finite amount of N_2 can be transported by a given quantity of blood, and if the ambient pressure is reduced too rapidly, the tissue nitrogen cannot be transported rapidly enough, and the formerly nitrogen-saturated tissues become supersaturated, with the result that bubbles form in the tissue.

The bubbles may form in any tissue including the blood. The place of appearance of the bubble and its size determine the specific type of pathology and thus the resulting symptomatology.

Trapped Gases.—Etiology and symptomatology of hypobaric effects on humans due to trapped gases result from their expansion due to the decreased barometric pressure of ascent to altitude and the resulting pressure phenomena on the involved viscera and adjacent organs. Effects on the sinuses, ears, and teeth are covered in detail elsewhere. The only trapped gas effects to be discussed in this chapter are those due to expansion in the hollow abdominal viscera.

Presdisposing Factors

Any cause of increased tissue nitrogen tension or decreased excretion rate may predispose to dysbarism when ambient pressure is reduced by low pressure chamber or aircraft ascent. Conversely, a rapid ascent may cause dysbarism in the face of normal tissue P_{N_2} and normal excretion rates. The whole matter is made much more difficult by the existence of marked individual variance in susceptibility to hypobarism, and even differences in the same individual from one exposure to another. Some air forces make a trial exposure to hypobaric conditions a matter of selection for aviation training by utilizing a standard stimulus and rejecting those individuals who become symptomatic. This is not routinely done in the Navy.

Any individual can be forced to experience dysbarism by making a rapid ascent to high altitude and then exercising. The purpose of this section, however, is to present the factors which may facilitate the appearance of dysbarism without such aggravation.

Age.—While it is generally agreed that the risk of dysbarism increases with advancing age, there is certainly not a direct relationship. There is instead an increased statistical frequency. However, as age increases so does weight, and the muscle/fat ratio changes also. Such generalities cannot be used to specify risk in a particular case, but only in age groups. Table 11–16 shows the relationship between age and susceptibility to evolved gas hypobarism.

TABLE 11–16. *Relationship Between Age and Susceptibility in Subjects Exposed to 28,000 Feet for 2 Hours*

Age group	Total number	Number with symptoms	Percentage susceptible	Statistical significance*
17–20	642	5	0.78	(§)
21–23	599	10	1.67	(‡)
24–26	482	24	4.98	N.S.
27–29	296	22	7.43	(§)
30–35	384	23	5.99	(†)
36+	230	12	5.22	N.S.
Total	2,633	96	3.65	

* Significance of difference from mean susceptibility at level of probability. † 0.05. ‡ 0.01. § 0.001.

(Fryer & Roxburgh, 1965, by permission of the Controller of Her Britannic Majesty's Stationery Office.)

Obesity.—Fatty tissue is less vascular than muscle tissue. Nitrogen transport, therefore, is slower from fat tissue to blood than from muscle to blood. In addition, nitrogen is five times more soluble in fat than in water. Thus, at equilibrium, fatty tissue has five times more nitrogen than blood or muscle. The combination of large stores and poor transport causes dysbarism to occur in fatty tissue much more frequently than elsewhere. Generally speaking, the more obese the aviator, the greater the probability of dysbarism.

Exercise.—At altitude, exercise definitely increases incidence of dysbarism. Figure 11–15 shows the relationship between duration of exposure, amount of exercise, and symptom incidence. The reason for this changed incidence is not definitely known but is thought to be due in part to the increase in tissue metabolic CO_2 incident to the exercise. Exercise causes a higher incidence of symptoms occurring at a lower altitude and after a shorter time duration than that experienced at rest.

Rate of Ascent.—Haldane's work (1907) showed that stepwise ascent from depth (in water) was feasible, and that it was possible to decompress a man directly to a point at which the total pressure was not less than half the pressure of dissolved nitrogen in the tissues. Therefore, rate of ascent within this limit would theoretically have no effect on incidence of bends, but rate of ascent outside the limit would. Although these observations are the very foundation of divers' decompression tables, their applicability to aerospace medicine is open to some question. Indeed, Fryer and Roxburgh (1965)

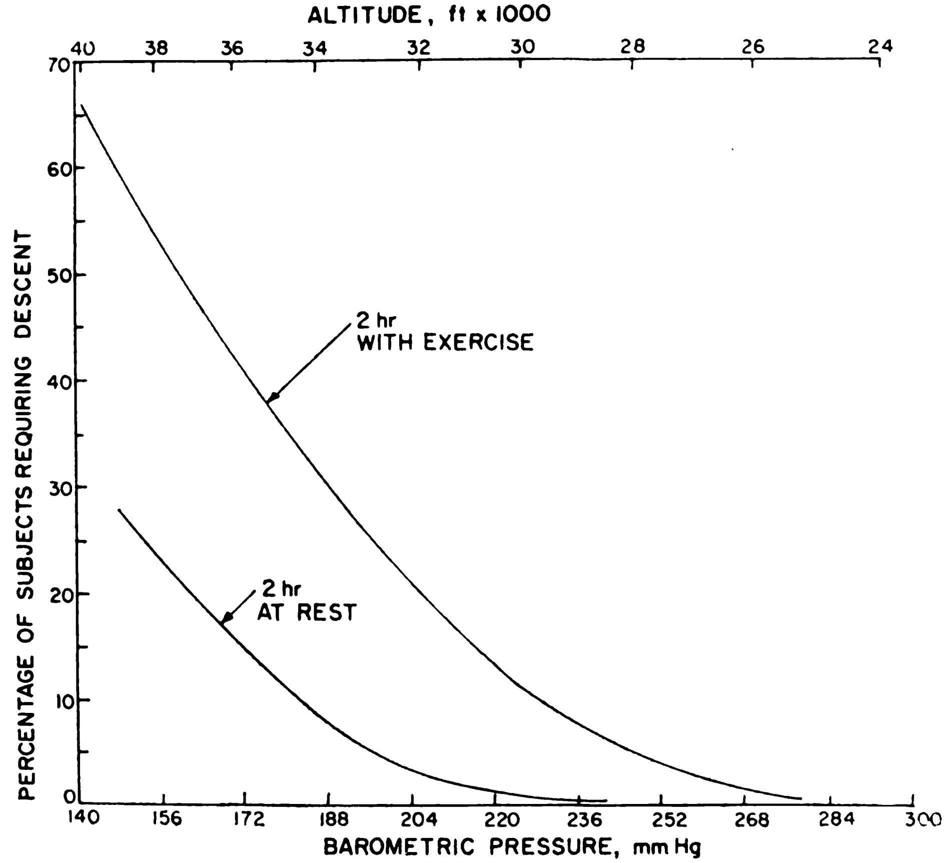

FIGURE 11-15. *Comparison of the incidence of decompression sickness during exposure to various altitudes for two hours with and without exercise.* (FRYER & ROXBURGH, 1956, BY PERMISSION OF THE CONTROLLER OF HER BRITANNIC MAJESTY'S STATIONERY OFFICE.)

feel that rate of ascent is *not* a causative factor and that it should be kept high to minimize the time duration of exposure to decreased barometric pressure. During ascent, breathing 100 percent oxygen, denitrogenation is being accomplished, so that rate of ascent does have some effect.

Attained Altitude.—Figure 11-15 indicates the altitudes at which dysbarism becomes a problem. In the literature, it is relatively rare at altitudes below 25,000 feet, although Fryer (1964) has reported a case at 18,500 feet. Certainly, if the bubble theory is correct, the greater the altitude, the larger the bubble. It has been previously noted that at 18,000 feet, the barometric pressure is only half that at sea level and is, therefore, roughly at Haldane's upper limit.

Increased Tissue P_{N_2} *Prior to Ascent.*—The pressure changes involved in proceeding to various underwater depths are illustrated in table 11–17. The rate of increase in barometric pressure in water is extremely rapid when compared with the rate of decrease in the atmosphere. A descent of only 33 feet will cause the barometric pressure to be doubled to two atmospheres, whereas an ascent from sea level up to the edge of the earth's atmosphere at 200,000 feet changes the barometric pressure by less than one atmosphere. A descent of only 132 feet will produce a fivefold increase in pressure.

TABLE 11–17. *Pressure Changes at Various Diving Depths*

Depth (feet)	Absolute pressure			O_2 partial pressure		N_2 partial pressure	
	atm.	psi	mm Hg	psi	mm Hg	psi	mm Hg
Surface	1	14.7	760	2.94	152	11.76	608
33	2	29.4	1,520	5.88	304	23.52	1,216
132	5	73.5	3,800	14.7	760	58.8	3,040

(*U.S. Navy Diving Manual* 1963.)

When breathing air at these increased pressures, the partial pressures of the constituent gases show a comparable increase. For example, at a depth of 100 feet (4 atmospheres), 5 percent oxygen is equivalent to 20 percent oxygen at sea level. However, such a P_{O_2} probably would not be used inasmuch as ascent would rapidly render the individual hypoxic unless the P_{O_2} were increased in direct proportion to the pressure decrease.

Similarly, two percent carbon dioxide in a mixture inspired at 132 feet (5 atmospheres) will produce the same physiological effects as 10 percent carbon dioxide inspired at sea level, i.e., it will generally cause unconsciousness. Therefore, all metabolic CO_2 must be vented overboard or absorbed in underwater work.

Most importantly, the effect of increased barometric pressure on nitrogen must be considered. When a person resides at sea level, his blood and all tissues become saturated with dissolved nitrogen at a tension equal to $P_{N_2(alv)}$. With an increase in barometric pressure, the $P_{N_2(alv)}$ increases and the saturation level of blood and tissue increases proportionally according to Henry's Law. For example, in an exposure to a depth of 132 feet (5 atmospheres), breathing air, the total nitrogen in solution in the tissues will be 5 times as great as that at one atmosphere if sufficient time is allowed to achieve saturation (up to 12 hours). In concrete terms, this means about 5,500 to 7,500 milliliters of dissolved nitrogen in the tissues at saturation. When the individual begins an ascent, it must be undertaken in steps according to Haldane's formulas (1907) and more recent Navy diving tables (*U.S. Navy Diving Manual,* 1963), or else must be performed so slowly as to be impractical. If the ascent is performed in any other way, the tissues reach supersaturation with nitrogen, due to the inability of normal respiratory washout to keep pace with the large quantities of evolved gaseous nitrogen. Bubbles then form and dysbarism occurs in its classic forms.

Scuba Diving Versus Altitude Dysbarism.—Of particular interest to Flight Surgeons is the relationship between scuba diving and susceptibility to dysbarism during high-altitude flight. Dysbarism is infrequent below altitudes of 20,000 feet. However, an instance in which several members of the crew of a civilian airliner developed disturbances at an altitude of 7,000 feet has been recorded (*Approach,* 1963). Investigation showed that all had been scuba diving immediately before the flight. For this reason, the Military Airlift Command requires all aircrewmembers participating in scuba diving to a depth below 15 feet within 12 hours of a flight to obtain medical clearance if they are participating in flights with cabin altitude in excess of 5,000 feet.

The Navy rule for evaluating physiological safety for actual flight following scuba diving is somewhat different: "The general rule of 12 shall be applied: no flight above 12,000 feet for 12 hours following a scuba dive of 12 feet or more."

The physiological reason for this rule is that enough time must be allowed at sea level, breathing air, to complete respiratory washout of the excess nitrogen stored in the tissues during the dive. "Excess" is defined as that amount present over and above that required to produce nitrogen saturation at one atmosphere.

Decreased P_{N_2} *Prior to Ascent.*—Experimental work on inhabitants of mountain villages (Hurtado, 1964) has shown that they have far less incidence of decompression sickness than persons dwelling at sea level for similar ascents, probably due to the decreased tissue P_{N_2}. The technique for utilizing this fact is denitrogenation, which is covered in detail later.

Other Factors.—Other factors affecting altitude dysbarism are:

1. Previous injury—bubbles tend to form at sites of recent injuries in some cases.

2. Repeated exposure—some individuals who have once had an attack of dysbarism may be more susceptible to another such attack.

3. Total dissolved gas tension has been implicated as the cause of dysbarism, rather than simply nitrogen tension. This would account for the increased incidence following exercise but leaves other questions unanswered.

4. Dysbarism occurs with greater frequency at lower ambient temperatures, possibly due to decreased nitrogen transport associated with peripheral vasoconstriction due to cold.

Clinical Features

Hypobarism (Evolved Gas Type).—Bends is defined as a clincial manifestation of hypobarism consisting of pain referred to the joints, muscles, and long bones. It customarily appears after an ascent but usually after a lag period of variable length. The site of appearance is most often the knee, followed by the shoulder, then the elbow, wrist, hand, and fingers in descending order of frequency. The pain may become worse or better with time and may even disappear. This is the most frequent type of evolved gas hypobarism. Bends is not considered a serious condition per se and is usually relieved by descent. The theoretical causal mechanism of bends is formation of nitrogen bubbles in periosteal vessels; in intra-articular fat and fluid, and in the muscle and fascial planes.

Chokes is defined as a form of hypobarism characterized by substernal distress, nonproductive cough, and difficulty in breathing, accompanied by a sense of apprehension and suffocation. Chokes is a much more serious condition than simple bends. Theories of precise etiology are numerous and probably all have some basis in fact. However, almost all the symptoms can be explained as being due either to the formation or collection of bubbles in the pulmonary capillaries, and/or to the effects of extravascular mediastinal bubbles exerting pressure effects on mediastinal contents and adjacent pulmonary tissue. Chokes may occur as the only symptom, but most often is associated with bends. When an individual develops both symptoms, the chokes usually appear later in the flight than the bends. Chokes tend to progress more than bends and to be more disabling.

Cough and substernal distress in chokes are both markedly aggravated on attempting to take a deep breath, which results in decreased pulmonary ventilation and thus hypoxia. It is not surprising, therefore, that syncope and collapse are more frequent than in bends. In fact, onset of chokes should be regarded as an emergency requiring immediate recognition and action, consisting of prompt descent and termination of the low pressure chamber or aircraft flight, and appropriate management as outlined below.

Central nervous system disorders are another form of hypobarism. Bubbles formed anywhere in the body can lodge in the arterioles or capillaries of the brain, causing almost any central nervous system disturbance imaginable, depending on bubble size and location. Secondary vasospasm may occur, resulting in even greater severity of manifestations. The usual central nervous system symptoms are referable to the senses, especially vision, and to disturbances in orientation. *Any central nervous system symptom is an indication for mandatory hospitalization.* Nevertheless, most, if not all, symptoms of central nervous system dysbarism disappear on the recompression incident to descent to sea level. The reason for hospitalizing all aviators or low pressure chamber personnel who have had an attack of central nervous system hypobaric effects is that at various unpredictable times, after apparently complete recovery, sudden and severe neurocirculatory collapse may occur, *with no warning.* The incidence of central nervous system symptoms is low, and the effects are temporary, but a high level of awareness is needed by the Flight Surgeon in dealing with all inflight incidents. Otherwise, the temporary visual defect which disappears on descent, or the staggering gait which may be the only effect noted will be assigned to other causes or summarily dismissed.

Hypobaric collapse is discussed by Clamann (1961) in Armstrong's *Aerospace Medicine.* Clamann states that in about 1.0 percent of severe bends and 25 percent of chokes, syncope occurs. He further states that shock may result from the syncope and that neurocirculatory collapse may ensue. Little is known about hypobaric collapse, except that it does occur, is usually preceded by bends or chokes, is more frequent later in the flight (occurring usually after bends and/or chokes), frequently follows central nervous system manifestations, and may have no premonitory signs or symptoms at all.

As an example, a rather obese chief petty officer noted visual illusions consisting of "bright spots before the eyes" during a chamber flight, with no other complaints or findings. Immediate descent caused alleviation of all symptoms, and the individual was quite hostile to the idea of hospitalization. He was transported to the hospital lying down, protesting all the way, and was admitted with no presenting complaint. About one hour after admission and two hours after the original complaint of mild visual illusion, the patient suddenly went into profound shock with no obtainable pulse or blood pressure. Emergency therapy was ready at the bedside for just such an eventuality, and the patient recovered after an agonizing half hour of treatment. He was observed for 48 hours thereafter and then discharged with no complaints. Such a reaction in an aircraft or in the home might well have proved fatal.

Skin manifestations may result when nitrogen bubbles occurring intra- and extravascularly cause pressure phenomena on dermal nerve endings, adjacent nerves, and blood vessels. Subdermal emphysema is a frequent occurrence and is easily identified by the crepitation on palpation. Formication is frequently reported, along with skin rashes, mottling, itching, burning, skin paresthesia and anesthesia. These manifestations may occur as the first sign of evolved gas hypobarism, but usually disappear without sequelae on descent. They do, however, represent warning of bubble formation and therefore are a danger sign.

Hypobarism (Trapped Gas Type).—Gas trapped in a hollow viscus will expand when the ambient pressure is decreased, doubling its volume at 18,000 feet and quadrupling it at 33,000 feet. The usual locations causing difficulty are stomach, small intestine, and colon, including rectum.

When gas is in the stomach, belching usually brings immediate relief, but if the gas pressure is not removed or reduced, a diaphragmatic herniation may be produced.

In the colon, gas expansion due to ascent produces the usual symptoms of flatulence and expulsion of the gas brings immediate relief. If for any reason the gas is not expelled, the symptoms of colonic distention will be noted if the pressure is decreased sufficiently or if the amount of gas is sufficiently large initially. Such symptoms can even become a coliclike pain leading to a generalized vasomotor reaction characterized by pallor, sweatiness and syncope, but such reactions are unusual due to the use of full pressure suits and/or pressure cabins.

In the small intestine, trapped gas reacts in a similar way, but is difficult to remove by either route. To make matters worse, gases in solution in the intestinal contents tend to come out of solution when the ambient pressure is decreased, thus adding evolved gas to the trapped gas in the intestine.

Hyperbarism.—Barotalgia and barosinusitis are covered in chapter 7.

Differential Diagnosis

Evolved gas hypobarism occurs after an ascent, whether from depth to sea level or from sea level to altitude. Therefore, a history of exposure is a prerequisite. Thereafter, many of the effects of hyperventilation, hypoxia, and evolved gas hypobarism overlap. Differential diagnosis between hypoxia and hyperventilation is given elsewhere. The major point in differentiating the chokes from hyperventilation is that deep breathing is almost definitive in hyperventilation and hypoxia whereas it aggravates the chokes and therefore is not seen. The chokes may be differentiated from acceleration atelectasis by X-ray and by history of onset of the first, following simple ascent to high altitude, while the other usually follows an acceleration-producing maneuver at lower altitudes. Other differentiations are much more difficult because the basic pathophysiology in evolved gas hypobarism is tissue hypoxia due to arterial occlusion by bubbles. The differential would appear to be easy when recompression causes immediate and complete relief of symptoms, but recompression also brings increased Po_2. Therefore, the differential must be considered as one of exclusion. If the incident occurred after an exposure to an altitude of 20,000 feet or higher, and if the O_2 supply was not interrupted or insufficient, a presumptive diagnosis of evolved gas hypobarism may be made.

Bends may be confused with muscle or joint strain. Usually, external evidence of trauma would tend to rule against bends, but conversely bends is known to occur more frequently in recently injured joints. The acid test in this case is recompression to sea level which will almost always alleviate the bends pain but will have no effect on the injury pain.

The pain of trapped gases may be confused with that of an acute surgical condition of the abdomen. If expulsion of gas relieves the pain permanently, it may be considered that trapped gas hypobarism was the cause. However, if expulsion brings only partial relief, descent and examination are necessary to make the differential diagnosis. Generally speaking, pain or neurological disturbances of any type are sufficient reason to descend to a lower altitude and in the majority of cases to terminate the aircraft or chamber flight.

Clinical Management

Evolved Gas Hypobarism at the Low Pressure Chamber.—One of the primary responsibilities of a Flight Surgeon is to provide medical support during low pressure chamber runs and to administer emergency treatment in the event of a decompression reaction on the part of persons within the chamber. The *Utilization Handbook for Device 9A9* lists procedures to be followed by chamber operating personnel and the Flight Surgeon.

Prior to all chamber flights, the station dispensary is notified that the low pressure chamber will be in operation. This information is passed on to the duty Flight Surgeon, who is then able to observe the actual chamber flight or is, at least, alerted that such training is in progress. During the chamber flight, participants should be cautioned to report any unusual symptoms immediately to the inside instructor. In the event of a chamber reaction, the duty Flight Surgeon is called upon to render assistance.

The initial treatment of all severe decompression reactions is recompression or descent. If in doubt as to diagnosis, descent is still indicated. The rate of descent has to be governed by each individual case and will depend largely upon the severity of the reaction. If the reaction is mild, a gradual descent is permissible, although if above 30,000 feet, descent to about 20,000 feet should be made at free-fall rates. If the reaction appears to be serious, free-fall descent to ground level may be indicated in spite of discomfort to other occupants. However, one must not fail to observe all other occupants. In all cases of collapse, a horizontal position with feet elevated is desirable. Always insure free airway and administer 100 percent oxygen throughout the descent. If apnea exists, artificial respiration is, of course, mandatory.

All personnel who experience altitude chamber reactions must be referred immediately to a Flight Surgeon for postflight examination and disposition. The Flight Surgeon must remember that most persons suffering mild chamber reactions will have no sequelae. However, if a bubble remains, even though decreased in size, its effects on circulation will be detectable on physical examination. Central nervous system effects noted during a chamber run make hospital admission mandatory *in every case,* as a precaution.

Evolved Gas Hypobarism While Airborne.—NATOPS Manual OPNAV-INST 3710.7 series states, "When it is observed or suspected that an occupant of any aircraft is suffering the effects of decompression sickness, the pilot will immediately descend, land at the nearest installation, and obtain qualified medical assistance. The person affected may continue the flight only on the advice of a Flight Surgeon." The Flight Surgeon will act as he feels indicated in the individual case. Some aspects of management are covered in the next section.

Evolved Gas Hypobarism, Postflight.—As with hypoxia, most of the symptomatology is alleviated by descent. An occasional case will continue to have complaints and physical findings. One must remember that confusion and disorientation are prominent findings in cerebral hypoxia whether caused by low P_{O_2} (hypoxic hypoxia) or by occlusion of the arterial blood supply (stagnant hypoxia) by bubbles. Therefore, any person even suspected of having suffered decompression sickness (evolved gas hypobarism) should have a thorough history (including mental status) taken, preceded by a physical examination to include a thorough neurological examination. The inverse order of examination, followed by history taking, is necessary to detect residuals before they disappear. The symptomatic case should be treated differently, however, and should be immediately placed in a recompression chamber, if available. If the patient is unable to speak coherently, another crewmember should be questioned as to the facts of the flight and onset of the disease while treatment continues. Immediately required facts include time of takeoff, rate of climb, attained altitude, stay time at altitude prior to onset of disease, changes in cabin pressurization, precise and inclusive symptoms noted, any transient paralyses or other signs of neurological involvement, and disturbances of consciousness.

Any individual showing any central nervous system symptoms, such as the bends, even though all symptoms may have subsided, should be admitted to the hospital for at least 24 hours and observed closely during this period. There may be a delayed onset of severe neurocirculatory collapse. Even though the examining physician finds the subject to be symptom-free, he should be hospitalized for this period.

Diagnosis should be based on:

1. History of exposure.
2. Symptoms suggestive of neurologic involvement, such as numbness, paresthesia, and motor weakness. In severe cases, cerebral symptomatology and coma may predominate.
3. Vascular instability, and syncope on standing, which should alert the physician to hypovolemic changes. Blood pressure usually is maintained for some time after much plasma is lost. Shock has a sudden onset in these cases.

The following laboratory and other baseline data should be secured:

1. Body weight.
2. Hematocrit.

3. Hemoglobin.
4. Complete blood count.
5. Plasma volume and red cell volume.
6. Serum electrolytes.
7. Electroencephalogram.
8. Electrocardiogram.
9. Intake and output. Establish a strict record to include an hourly urinary output through an indwelling catheter.

Malette et al. (1962) present suggested treatment procedures for dysbarism; while prefacing the listing of procedures with the caution that "a typical case of dysbarism presents a mixture of neurologic and hypovolemic symptoms. A high degree of clinical judgment is needed to assess the predominant lesion. Much critical analysis will have to precede intelligent therapy in each case. Proper weight must be given each symptom and finding in the estimate of the clinical situation and therapy directed in a logical manner." They recommend the following protocol in the management of altitude dysbarism of the central nervous system:

1. Establish a venous fluid pathway.
2. Calculate static plasma deficit and begin replacement of plasma volume. (Avoid vasopressors.)
3. Maintain urinary output of 35ml per hour, as minimum.
4. After initial plasma replacement, repeat hematocrit and recalculate deficit for continuing loss.
5. Follow patients with frequent checks of respiration and breath sounds.
6. As hematocrit approaches normal levels, begin balanced administration of fluids other than plasma to cover intake and output.
7. As diuresis begins and urinary output increases above 50ml per hour, start daily maintenance fluids. (By this time, the patient may be maintained on oral intake.)
8. Maintain strict bedrest until patient gives both clinical and laboratory evidence of recovery.

Coburn, Gould, Young, Hatfield, Colley, and Martin (1962) point out that hospital medical staffs are usually ill prepared to deal with collapse due to evolved gas hypobarism, and that it is both fairly rare and quite serious. It behooves the Flight Surgeon to be aware of the disordered physiology, diagnostic aspects, and what little is known of therapeutics for this condition.

Decompression Therapy

In instances of mild decompression sickness, the symptoms typically are promptly and completely relieved by recompression to sea level atmosphere. In more serious cases, however, the reaction continues unabated when the individual is returned to ground level pressure. If the episode is due to the release of nitrogen bubbles, the question then arises as to why descent to ground level does not in these instances cause the bubbles to go again into solution. Downey, Tracy, Hockworth, and Whitley (1963) studied this question by experiments with human serum in a small pressure chamber. Pressure above the serum was reduced to an altitude equivalent to 43,000 feet. After bubbles had evolved at altitude, the barometric pressure was brought back to ground level. The bubbles markedly decreased in size, but did not disappear. The chamber atmospheric pressure then was further increased to 75 psi. At this level, the bubbles had decreased much more in size, with many of them disappearing altogether. This study obviously provides a rationale for the use of overcompression in the treatment of dysbarism.

Donnell and Norton (1960) report the successful use of the compression chamber in treatment of an instance of severe decompression sickness with neurocirculatory collapse. In this instance, the patient who was moribund, was placed in the compression chamber approximately five hours after the initial onset of symptoms. He was subjected to a pressure of six atmospheres, corresponding to a depth of 165 feet of sea water. On reaching this pressure, 14 minutes after leaving the surface, the shock state began to disappear. Blood pressure improved and stabilized at 110mm Hg systolic pressure. The patient was retained at this pressure level for two hours and then gradually was decompressed over a 38-hour period. During this prolonged slow decompression, improvement was gradual but sustained. Upon leaving the chamber, recovery was virtually complete.

Goodman (1964) provides a report of 14 successful cases of treatment of dysbarism through overcompression. In this report, it is suggested that the successful clinical experience with compression is itself an etiological argument for the bubble hypothesis of dysbarism reaction. Goodman also feels that the experimental observations of bubble formation, combined with the successful clinical experiences seem to advocate the early institution of additional applied compression, and to condemn purely supportive regimen.

Based on the 14 successful experiences, the following conclusions are drawn:

1. The sooner that recompression is instituted after an episode, the better are the chances for complete recovery.
2. With a delay of two hours or longer before compression, use of Navy Treatment Table 3 or 4 (*U.S. Navy Diving Manual,* Table 1–21, 1963) will ultimately be followed by recurrences and/or residual effects.
3. In severe dysbarism, with delayed institution of compression therapy, the use of oxygen is indicated.
4. For cases not notably complicated, the depth of compression required for symptom remission does not exceed three atmospheres absolute.

Table 11–18 summarizes specific situational data for patterns of therapeutic exposures which are felt to be both optimal and safe.

Trapped Gas Hypobarism (*Boyle's Law*).—If pain or other severe symptoms occur, and cannot be relieved by expulsion of the gas one way or another, descent from altitude will alleviate the difficulty. Mission aborts for such reasons can be best treated by prevention.

Frequent causes of intestinal gas distention are: dietary factors, aerophagia, and intestinal upset.

Dietary factors known to be important in producing abdominal distress at altitude are gas-forming foods, foods which contain gastrointestinal irritants, and foods which produce allergic reactions for particular individuals. In general, it has been found that high-carbohydrate meals are more likely to increase gas volume than high-protein meals. Melons, carbonated water, and beer produce gastrointestinal bloating at altitude.

Gastrointestinal irritants may include spicy foods, condiments, onions, beans, cabbage, peanuts, peppers, and cucumbers. Such foods do not necessarily contribute to the gas volume but do produce increased abdominal distress through alterations in the sensitivity and motility of the intestinal tract.

Any cause of *aerophagia* must be avoided. Therefore, aviators should have the opportunity to eat a leisurely meal in quiet, relaxed surroundings prior to a flight. Chewing gum should be avoided both before and during flight.

Any *gastrointestinal upset* causing diarrhea, nausea, or vomiting may also produce larger quantities of intestinal gas than normal and may interfere with normal expulsion. For this reason alone, aviators with intestinal upsets should be grounded until definitely recovered. Another excellent reason is that electrolyte disturbances which are commonly associated with gastrointestinal irritability may cause severely decreased capability in reacting to stress

Table 11–18. *Recommended Compression Profiles for Normal Recoveries and for Those With Persisting Symptoms*

A. DEPTH-TIME PARAMETERS FOR MINIMAL COMPRESSION-OXYGEN BREATHING THERAPEUTIC PROFILE

Treatment phase	Depth (feet)	Time (minutes)	Total time (minutes)	Gas	Remarks
Compression	0–60	2	2	Oxygen	Rate variable.
Test of relief	60	10	12	Oxygen	Must be complete; use table B if symptoms persist.
Relief complete within 10 minutes	60	26	38	Oxygen	
	60	2	40	Air	
Ascent to 33 feet	60–58	2	42	Air	Uniform ascent, 1 FPM.
	58–33	25	67	Oxygen	
1 atmosphere absolute	33	30	97	Oxygen	Uninterrupted.
Final ascent	33–0	33	130	Oxygen	Uniform ascent, 1 FPM.
B. METHOD FOR PERSISTING SYMPTOMS FOR MAXIMAL, SAFE TISSUE REOXYGENATION					
Compression	0–60	2	2	Oxygen	Rate variable.
Breathing mixture alternation pattern for 60-feet exposure	60	28	30	Oxygen	Interspersed air breathing prolongs OHP preconvulsive latency.
	60	15	45	Air	
	60	28	73	Oxygen	
	60	2	75	Air	
Ascent to 33 feet	60–58	2	77	Air	Uniform ascent, 1 FPM.
	58–33	25	102	Oxygen	
Breathing mixture alternation pattern for 33-feet exposure	33	15	117	Air	Interspersed air periods for comfort; (attendants on air entire 285 minutes).
	33	60	177	Oxygen	
	33	15	192	Air	
	33	60	252	Oxygen	
Final ascent	33–0	33	285	Oxygen	Uniform ascent, 1 FPM.

(Goodman, 1964.)

whether induced by acceleration, heat load, or the exertions of escape and evasion.

Such intestinal upsets are relatively frequent in foreign shore areas, especially in advanced Marine Corps bases under combat conditions. The Flight Surgeon must remain alert to the incidence and prevalence of such conditions because some aviators consider them unimportant and do not report to sick call. Occasionally a large proportion of a unit is on some sort of self-grounding before the Flight Surgeon becomes aware of the problem. By the same token, considerable effort in obtaining good food and water sanitation is justified, in order to maintain combat readiness of the aviators.

Prevention

Denitrogenation ("Nitrogen Desaturation" or "Preoxygenation").—A good method of prevention of evolved gas hypobaric effects is "denitrogenation" or removal of all dissolved gaseous nitrogen in the body. (Nitrogen is five times more soluble in fat than in water.) This amount of nitrogen represents saturation of the tissues under sea level pressures.

Denitrogenation is accomplished by establishing a "gradient" or nitrogen partial pressure difference between the tissues and the alveolar gas, such that alveolar P_{N_2} is low. Tissue P_{N_2}, therefore, diffuses readily to blood and thus to alveoli. If the exterior supply of nitrogen to the alveoli is kept low or nonexistent, and if the exhaled gas is vented overboard, eventually almost all the tissue nitrogen can be removed. The time required for complete denitrogenation is, however, about 12 hours, and the length of time required is the same regardless of the original gradient.

One limiting factor on nitrogen excretion is pulmonary washout rate. In the example given in Comroe (1965) of a man given 100 percent oxygen to breathe, the functional residual capacity was 3,000 milliliters; anatomical dead space 150ml, and alveolar ventilation 350 ml/breath. Each inspiration dilutes the nitrogen in the alveolar gas by 10 percent. The alveolar nitrogen, therefore, on the first respiration decreases from 80 to 72 percent. At the end of the second respiration, the alveolar nitrogen is 64.8 percent. This continues until alveolar nitrogen is washed out and the lung contains only O_2, CO_2 and H_2O vapor. The pulmonary nitrogen may be washed out in a few minutes, but tissue and, therefore, blood nitrogen are eliminated much more slowly because the nitrogen must diffuse from the tissue to the blood, be transported to the lung, and then diffuse from blood to alveoli, then be expelled from the lung on exhalation.

However, as tissue P_{N_2} decreases, the diffusion rate decreases since the gradient is decreased. In addition, blood supply to fatty tissue is rather low and tissue perfusion rates are correspondingly low. Therefore, the denitrogenation curve is not a straight line, as reference to figure 11–16 shows. Indeed, even after six hours breathing 100 percent oxygen to achieve the optimal gradient, nitrogen still remains in the tissue in small quantities.

Since denitrogenation for such time periods is out of the question for routine military aviation, some compromises must be made. Figure 11–17 shows the tradeoff between denitrogenation and bends incidence. Generally speaking, two hours denitrogenation will give maximum protection against evolved gas hypobarism, while one hour will give a considerable amount of protection.

The effect of preexposure denitrogenation is illustrated in figure 11–17. In all cases, after a control period at ground level subjects were taken to 38,000 feet breathing oxygen, at which altitude they performed five knee bends every three minutes until the appearance of joint pains, presumably caused by extravascular bubble formation.

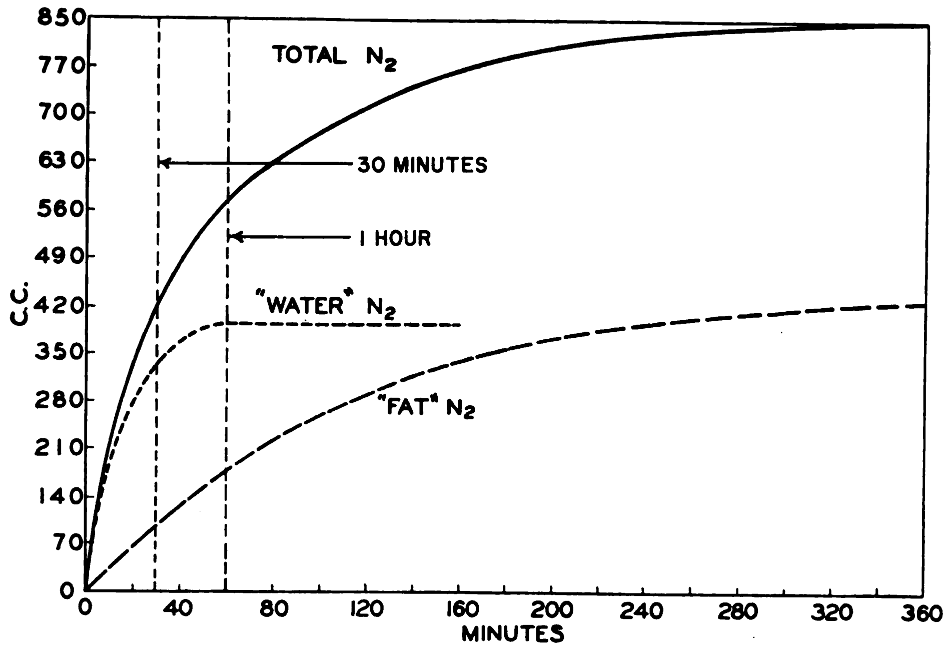

FIGURE 11–16. *The rate at which nitrogen is eliminated from the body at sea level when pure oxygen is breathed* (CLAMANN, 1961; DATA FROM BEHNKE).

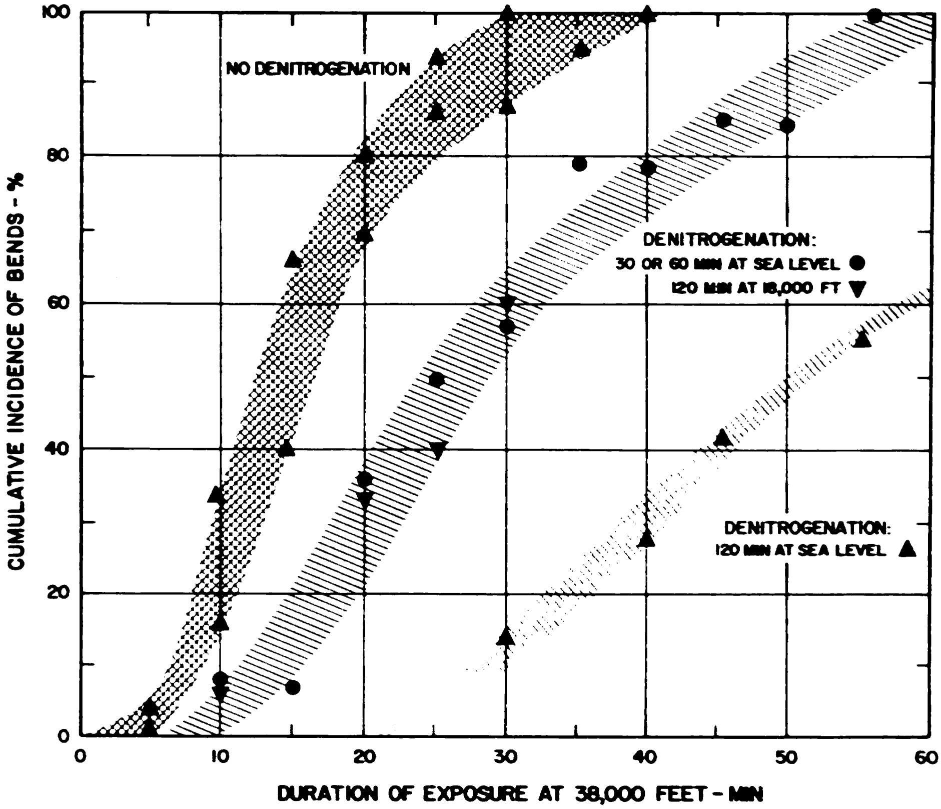

FIGURE 11–17. *Protection against decompression sickness.* (BILLINGS & ROTH, 1964; DATA FROM BALKE; & MARBARGER ET AL.).

The time periods noted apply only to continuous breathing of oxygen from time of beginning denitrogenation until time of exposure to low barometric pressure. If any "break" occurs during this interval, and the subject breathes air for a short period, the curves of figure 11–16, and the corresponding denitrogenation times are markedly affected.

A "break" of five minutes after one hour on 100 percent oxygen would require another 25 minutes on oxygen to return to the same state of denitrogenation. Similarly, after four hours of denitrogenation, a "break" of five minutes would require two hours additional oxygen breathing to return to the prebreak state. Even so, bends have been reported after long periods of denitrogenation, albeit rarely.

In the operational situation at present, purposeful denitrogenation is not utilized to protect against evolved gas hypobarism. One reason is that the pressure cabins and pressurization systems are so reliable and effective that a cabin altitude of 20,000 feet is rarely exceeded. If exposures to higher cabin altitudes are expected, the full pressure suit may be worn.

Denitrogenation, to be fully effective operationally, would require utilization of equipment not now in inventory. If the procedure were begun in the ready room of a carrier, the aviator would require a source of 100 percent oxygen in the ready room. After completion of denitrogenation and briefing for a total period of perhaps one hour without breaks, a portable oxygen source would be required to prevent a break while proceeding from ready room to aircraft. (Neither the ready room oxygen source nor the portable source is presently available at most stations nor is it likely that they will be.) The breath must be held both while transferring from ready room source to portable source, and from portable source to aircraft system.

Such an idealized procedure can always be improvised if the need for prophylaxis of evolved gas hypobarism becomes evident in a given situation. However, the aviator normally undergoes some denitrogenation in the normal course of events, although it is not especially planned that way. The first 100 percent oxygen breathing that the aviator performs is that which occurs from the aircraft system after he mans it. The period of oxygen breathing on the deck may be as long as 15 minutes, followed by that normal to ascent to the operating altitude. The ascent may add another 15 minutes, for a total of 30 minutes denitrogenation before reaching altitude. Reference to figure 11–16 shows that about 40 percent of the nitrogen stores of the body have been eliminated, and reference to figure 11–17 shows that a longer exposure time at altitude is possible for the same probability of experiencing bends than if a diluter-demand oxygen system had been utilized. An important fact is that very few attacks of bends occur after the first two hours of exposure.

At the low pressure chamber of the Aviation Physiology Training Unit, denitrogenation is routinely accomplished and is an effective means for preventing most cases of evolved gas hypobarism. In some cases, however, the hurried pace of training and investigation may cause inadequate durations of denitrogenation prior to beginning a chamber "flight." The Flight Surgeon must be on the alert for such violations of good chamber discipline and assist Aviation Physiologists in maintaining such discipline.

Pressure Cabins.—The simplest method of overcoming reduced barometric pressure is to provide sea level pressure conditions in the cockpit or cabin. In actual practice, however, the provision of sea level conditions becomes impractical for a number of reasons.

Reciprocating engine aircraft use engine drive compressors to provide pressurized air. Aircraft having gas turbine engines normally utilize "bleed" air from one of the compressor stages of the engine itself. The air supply must be capable of supplying an adequate volume of air during reduced engine power settings such as those associated with descent. Pressurization capacity thus must account for variations in power settings, decreased density of air at higher altitudes, and leakage rates from the pressure cabin. The higher the differential required between design cabin pressure and ambient pressure, the greater must be the capacity of the pressurizing system, and the stronger (and heavier) the fuselage construction. The pressurization of aircraft cabins represents an excellent example of engineering tradeoff. A high differential requires an aircraft structure which is physically stronger and therefore heavier than that required for a lower differential. The increased weight, in turn, decreases the payload of the aircraft. Pressurization requires an expenditure of energy; therefore, the larger the differential, the greater the power required to provide the desired pressure. The temperature increase which occurs when air is compressed must also be reckoned with. The higher the pressure, the higher the temperature, and heat exchanger capacity must necessarily be larger. The higher the pressure differential, the greater is the danger in the event of explosive decompression. Finally, cost also is a factor.

Therefore, the pressurization schedule selected for a particular aircraft is typically a compromise between physiological requirements, engineering capability, overall aircraft performance, and cost.

Commercial and military air transport aircraft typically maintain a cabin pressure equivalent to 8,000 feet of altitude (10.91 psi). Thus, flights to 40,000 feet (2.72 psi) are possible when the 8.5 psi differential usual to civil air transport design is maintained. For this type of operation, cabin pressurization to this level is entirely adequate. The slight loss in night vision capability which might be experienced does not appear to present any problem.

For military attack and fighter aircraft, other considerations are involved in the selection of an optimum pressurization system. The smaller cabin area of these aircraft means that loss of the pressure seal through material failure or enemy action will create a very rapid decompression.

In addition, rapid decompression may occur during the escape sequence for aircraft equipped with ejection seats. In these aircraft, the overhead canopy or escape hatch is explosively removed during the initial ejection sequence. Some systems actually use the ejection seat headrest as a hammer. During the upward movement of the seat the headrest contacts and fractures the plastic canopy, permitting the man/seat assembly to go through the canopy. Thus a rapid decompression is experienced.

In view of the above decompression possibilities, plus the fact that normal oxygen breathing equipment is adequate up to 43,000 feet and decompression sickness is unlikely at cabin altitudes below 18,500 feet, lower pressure differentials are used in military combat aircraft. Thus lower weight penalties are incurred, resulting in greater range and service ceiling.

H. H. S. Brown (1965)[1] summarized the merits of high and low cabin pressure differentials as follows:

High Differential

Physiologically more desirable cabin conditions are ensured.

The need for continued use of oxygen breathing equipment is avoided.

Feeding and toilet arrangements are simplified.

Moving about within the aircraft in flight presents fewer difficulties.

Low Differential

The physical risks attendant upon a sudden failure of pressurization are significantly reduced.

Since normal cabin altitudes require the constant use of oxygen equipment, occupants are in large measure already prepared to cope with the emergency of a pressure failure.

The rapid failure of cabin pressurization may be accepted as an operational hazard and crews can safely be given practical training in dealing with it.

Figure 11–18 shows a typical pressurization schedule found in naval aircraft. From sea level to 8,000 feet no pressurization takes place. From 8,000 feet to approximately 23,000 feet is an isobaric range in which pressure provides an 8,000-foot equivalent altitude. From 23,000 feet to the service ceiling of the aircraft a 5 psi differential is maintained. The NATOPS Fight Manual for each aircraft contains a description of the air-conditioning and pressurization system for that aircraft.

The air-conditioning and pressurization system of pressurized cabin aircraft controls the following:

1. Pressurization.
2. Ventilation.
3. Temperature.
4. Humidity.

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[1] By permission of the Controller of Her Britannic Majesty's Stationery Office.

The *pressurization system* consists of an air intake, a means of increasing the pressure of the air, and control mechanisms which maintain the desired pressure. As previously mentioned, in piston engine aircraft, air is obtained from ram air scoops, while the source for gas turbine-powered aircraft is bleed air from one of the engine compressor stages. For conventional aircraft, the air enters a rotary engine-driven compressor and is distributed to the cabin. The pressure differential is controlled by pressure sensors which in turn control the outflow of air from the cabin. For all practical purposes the pressurization controls can be classified in two categories. First are the type associated with transport aircraft in which the pressure control aneroid senses cabin pressure and maintains a constant pressure, e.g., 8,000-foot cabin altitude. The second type senses both cabin and ambient pressure and controls the cabin pressure on the basis of a fixed pressure differential, e.g., 5.0 psi. Combinations of these are found in most Navy tactical aircraft. Figure 11–18 shows a combination control with the first type maintaining isobaric cabin pressure until 23,000 feet is reached and the differential control taking over at higher altitudes.

Although pressurization is largely concerned with providing proper air pressure, other factors are important. High descent rates may produce severe aural barotrauma. In order to lessen this risk, some pressurization systems provide for a reduced rate of pressure increase during high rates of descent in the lower altitudes in which the rate of pressure increase is highest. For ex-

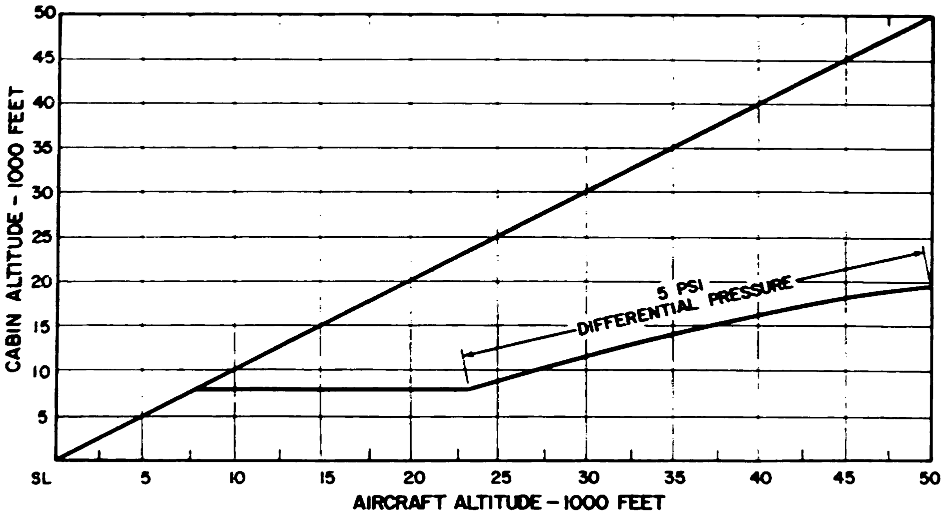

FIGURE 11–18. *Typical cabin pressure schedule for naval aircraft* (NATOPS FLIGHT MANUAL, NAVY MODEL A6A AIRCRAFT, APRIL 1964).

ample, when performing a jet penetration, the instrument procedures may require the pilot to descend at 4,000 feet per minute. The pressurization system may be limited to a 500-foot-per-minute rate of pressure increase below 8,000 feet. Thus, upon descending from 8,000 feet to 4,000 feet (in one minute) the cabin altitude will be 7,500 feet due to the deliberate lag built into the pressurization control device.

The pressurization system normally includes a cabin altimeter which permits the pilot to observe his cabin altitude. Controls also are provided to release cabin pressure and in some cases to select the cabin pressure differential. The latter capability permits the pilot to select a high pressure differential during cruise portions of a flight where the risk of loss of cabin pressure integrity through enemy action is minimal and to select a low pressure differential when in the high-risk combat area, thereby lessening the effects of a rapid decompression.

Ventilation is provided, to some degree, by the pressurization control system which normally regulates the amount of cabin leakage to maintain the desired pressure. The amount of air flow required to ventilate a cabin adequately has been recommended by McFarland (1953) as 35 to 40 cubic feet of fresh air per minute per passenger. In any event, 15 to 20 cubic feet should be fresh rather than recirculated air. McFarland also suggests a velocity of air movement of 20 to 60 ft/min. Brice (reported in Brown, 1965) recommends velocities of 25 to 40 ft/min when heating is involved and 40 to 50 ft/min when cooling of air is required. It is generally agreed that a feeling of stuffiness exists when air velocity falls below about 15 ft/min.

Military nontransport aircraft normally provide some flexibility in ventilation control at each crewmember station. Thus, foot ducts, seat cushion, head outlets, etc., can in many cases be selected and controlled by the seat occupant. Control is available for both the rate of flow of air and the desired temperature. In most cases, temperature is automatically maintained at the setting selected.

Temperatures, it is obvious, vary widely in the environments which one might encounter on the surface of the earth. These variations are a factor of both geographic location (and elevation) and time of year. Unfortunately, because of the compilation of so-called "standard" tables, it is not generally recognized that wide variations are also found at altitude. Figure 11–19, taken from H. H. S. Brown (1965), shows the variations in environmental temperature which can be encountered at different altitudes in the worldwide operation of aircraft. The center curve represents the values for temperature versus altitude depicted in the standard atmosphere of table 11–1.

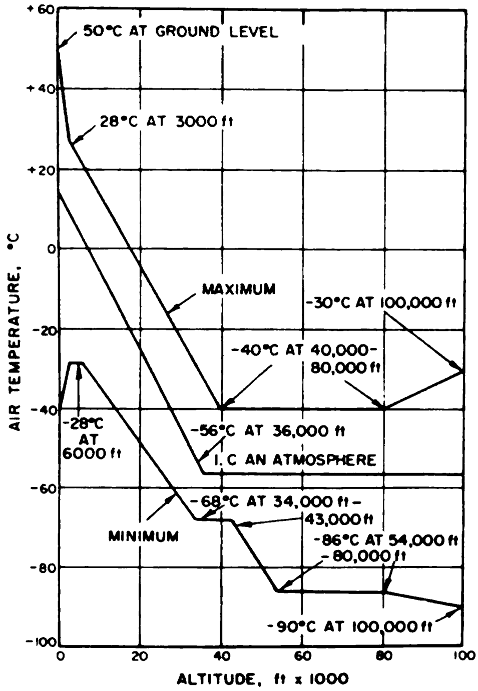

FIGURE 11–19. *Maximum and minimum atmospheric temperatures encountered in the worldwide operation of aircraft at various altitudes.* (BROWN, 1965, BY PERMISSION OF THE CONTROLLER OF HER BRITANNIC MAJESTY'S STATIONERY OFFICE.)

A number of factors influence the temperature of the aircraft cabin. High altitude flight is frequently above any cloud formations with the aircraft exposed to direct solar radiation. The transparent canopy and window materials permit the entrance of shortwave heat radiation, which is then reradiated about the cabin as longwave radiation to which the transparent materials are opaque. This greenhouse effect, plus the absorption of solar radiation by the airframe itself, causes an increase in cabin temperature.

As the aircraft travels through the air it tends to compress the air before it. While this effect is negligible at low airspeeds, it becomes a factor at high airspeeds. The increase in pressure is also attendant to an increase in temperature. This increase in temperature is frequently termed "ram rise" or simply "ram." Friction between the air and the aircraft also causes an increase in temperature. Both phenomena are termed collectively as "kinetic heating." Figure 11–20 indicates the rise in air temperature which can be expected at various airspeeds and at two air temperatures.

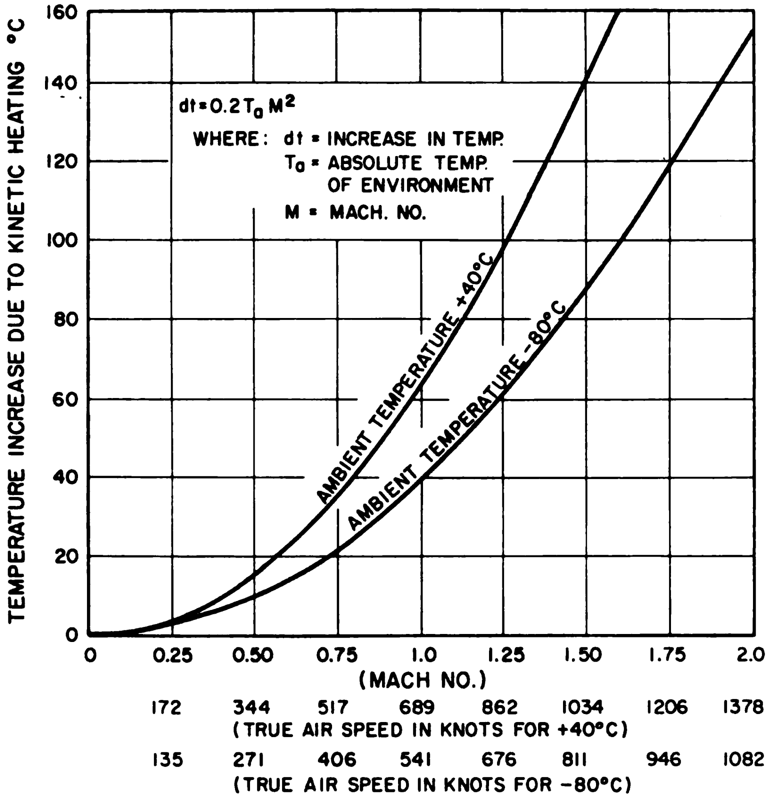

FIGURE 11–20. *Effect of airspeed and environmental temperature on kinetic heating.* (BROWN, 1965, BY PERMISSION OF THE CONTROLLER OF HER MAJESTY'S STATIONERY OFFICE.)

As mentioned, the compressing of air per se causes an increase in temperature. At lower altitudes where air is more dense the temperature rise is small and air must enter a heat exchanger to be warmed prior to being discharged into the cabin area. As altitude increases, the compressing of the less dense air causes a large rise in temperature which creates a need to cool the pressurized air prior to delivering it to the cabin. In table 11–19, Yaglou (reported in Brown, 1965) shows the effect of compression on the temperature of air while ascending to 45,000 feet and maintaining an 8,000-foot cabin altitude and a cabin temperature of 65° F. It is assumed that the aircraft climb true airspeed is 300 knots, that it has five occupants, for each there is an air supply of one pound per minute, cabin surface is 200 square feet. Inside wall temperature is 65° F and conductance of wall is 0.55 Btu's.

Table [illegible] Heating and Cooling Requirements of an Aircraft Cabin Kept at [illegible]° F and Pressurized to [illegible] Feet at All Altitudes

Plane altitude feet	Cabin altitude feet	Ambient temperature °F	Cabin heat loss Btu hr	Required temperature of air supply °F	Temperature of compressed air °F	Supplementary heating required Btu hr	Cooling required Btu hr
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	None
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	None
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	None
[illegible]	[illegible]	−67	[illegible]	[illegible]	[illegible]	[illegible]	None
[illegible]	[illegible]	−67	[illegible]	[illegible]	[illegible]	None	[illegible]
[illegible]	[illegible]	−67	[illegible]	[illegible]	[illegible]	None	[illegible]

From Bulletin [illegible] Decompression Sickness, Yaglou, C. P., Division of Medical Sciences, National Academy of Sciences—National Research Council, Washington, D.C., [illegible] pp., as reported in Brown, [illegible]

Absolute humidity, dew point, and ambient temperature decrease as altitude increases. When the cold, dry, ambient air found at altitude is compressed, some increase in humidity takes place. However, this is relatively insignificant. Table 11–20 shows the resulting relative humidity for various cabin differential pressures, assuming a final air temperature of 65° F and the starting air at maximum absolute humidity.

From the table it is obvious that additional humidification is required if a comfortable level of humidity is to be afforded passengers (30 to 50 percent). However, military tactical aircraft rarely provide any supplementary humidification. This presents no particular comfort problem since temperature control is available at almost each station and various flight garments are available to offset any chilling effects of low humidity. The drying effect of low humidity on oronasal passages presents a chronic but not serious problem.

Full Pressure Suits.—The full pressure suit may well be termed an extension of the aircraft pressurization system. The primary reason for the development of the full pressure suit was to insure a capability for mission completion under any eventuality. Should the aircraft experience loss of pressure, the mission can be continued at the assigned altitude if the pilot is wearing a full pressure suit even though flight above 50,000 feet might be required. In addition, the full pressure suit provides excellent protection during high-altitude ejection, as well as during the descent and during exposure to severe environmental conditions as might exist in a landing in the open ocean.

TABLE 11-20. *Effect of Altitude and Cabin Pressurization on the Relative Humidity Within Aircraft*

Aircraft altitude (feet)	Maximum absolute humidity of atmosphere (gm/gm)	Relative humidity in aircraft cabin for various cabin differentials, and assuming a final temperature 65° F								
		No pressurization (percent)	2 lb/in² (percent)	3 lb/in² (percent)	4 lb/in² (percent)	5 lb/in² (percent)	6 lb/in² (percent)	7 lb/in² (percent)	8 lb/in² (percent)	9 lb/in² (percent)
4, 000	0-0074	48.5	55.8	....	....	....	....	....	....	....
5, 000	0-0068	42.8	49.8	....	....	....	....	....	....	....
6, 000	0-006	36.4	42.5	....	....	....	....	....	....	....
7, 000	0-0055	32.1	37.7	40.6	....	....	....	....	....	....
8, 000	0-0049	27.6	32.6	35.1	....	....	....	....	....	....
9, 000	0-0043	23.3	27.7	29.9	32.1	....	....	....	....	....
10, 000	0-0039	20.4	24.4	26.4	28.5	....	....	....	....	....
15, 000	0-0022	9.45	11.75	12.85	14.0	15.1	16.3	....	....	....
20, 000	0-0012	3.96	5.46	6.05	6.7	7.3	7.95	8.55	....	....
25, 000	0-00056	1.585	2.17	2.46	2.75	3.04	3.34	3.61	3.91	4.2
30, 000	0-00024	0.545	0.792	0.92	1.04	1.16	1.29	1.415	1.53	1.67
35, 000	0-00006	0.108	0.17	0.201	0.243	0.263	0.295	0.326	0.358	0.388
40, 000	0-00006	0.085	0.147	0.178	0.21	0.341	0.272	0.303	0.334	0.365
45, 000	0-00006	0.067	0.129	0.16	0.192	0.223	0.254	0.285	0.317	0.347

(Brown, 1965, by permission of the Controller of Her Britannic Majesty's Stationery Office.)

The Navy full pressure suit (figure 11–21) was designed in terms of the following capabilities:

1. Provide breathing oxygen when needed during normal flight when a safe cabin pressure (3.4 psi absolute minimum) is maintained.
2. Provide breathing oxygen and maintain a minimum internal pressure of 3.4 psi absolute above 35,000 feet.
    (a) Using ventilating air and ship's oxygen supplied by the aircraft in the event cabin pressure is lost.
    (b) Using ship's oxygen supply, in the event both cabin and ventilating air are lost.
    (c) Using emergency oxygen supply in the event of ejection or loss of ship's oxygen supply and ventilating air.
3. Provide breathing oxygen only, using emergency oxygen supply in the event of ejection above 10,000 but below 35,000 feet.
4. Provide protection against sudden loss of cabin pressure (explosive decompression).
5. Provide protection from exposure to cold and water.
6. Keep pilot afloat in water, if face seal remains intact.

Figure 11–22 shows the pressure suit system schematic.

The full pressure suit handles the problem of explosive decompression in a unique way when compared with partial pressure suit garments.

During normal flight conditions the suit is ventilated with cabin conditioning air. Thus, while operating at 48,000 feet the cabin is pressurized to a 20,000-foot altitude. Ventilating air flows through the suit and the suit is at a 20,000-foot pressure. Upon the occurrence of an explosive decompression the suit controller immediately stops the outventing of ventilating air thereby momentarily holding the suit at 20,000 feet and then rapidly letting the ventilating air bleed down to 3.5 psia, equivalent to 35,000 feet. No time delay is present such as exists with partial pressure suits which exert a mechanical constriction to the body. The full pressure suit is always at or above the design pressure of 3.5 psia.

Indoctrination procedures given to persons upon initially being issued a full pressure suit (and during periodic reviews) include an actual explosive decompression in a low pressure chamber. This is done to instill confidence in the aviator regarding the protection which the garment provides.

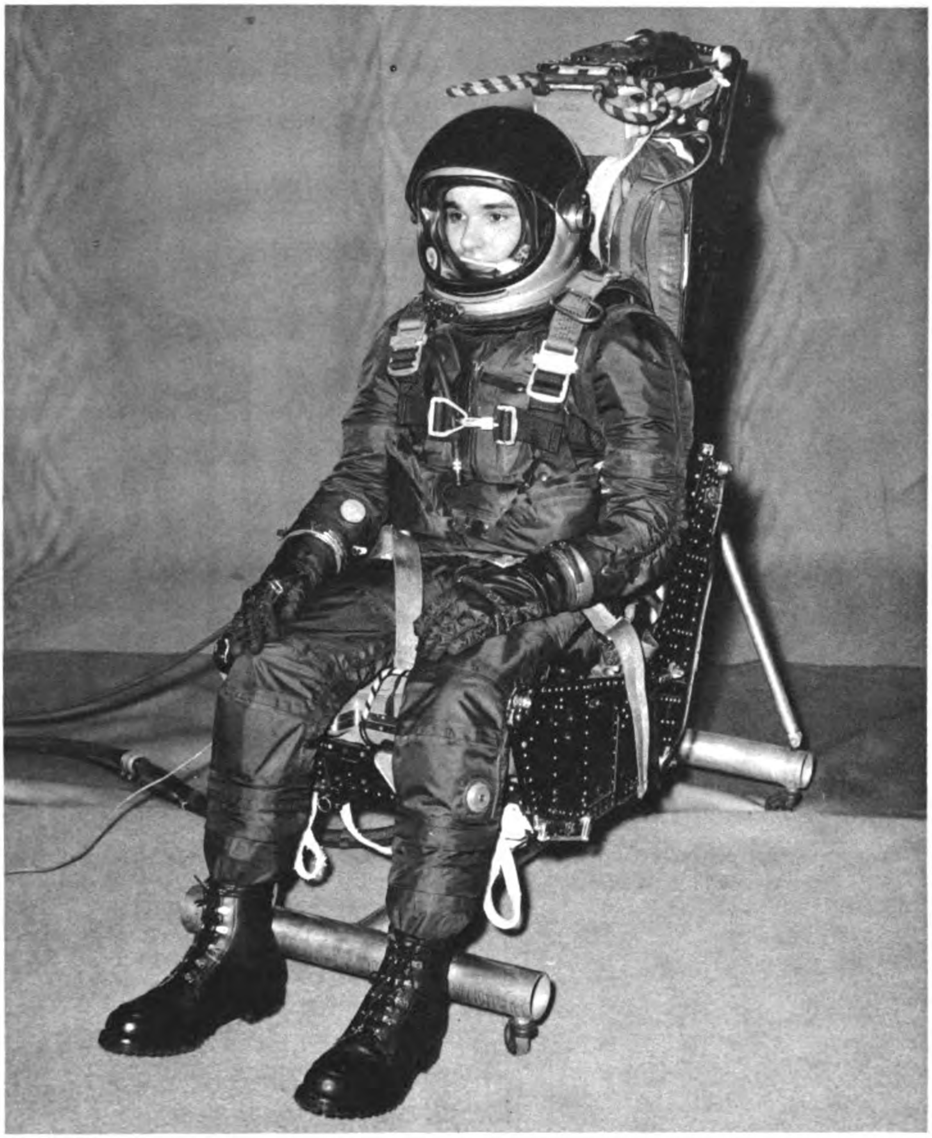

FIGURE 11–21. *Full pressure suit* (USN PHOTO).

Rapid and Explosive Decompression

When dealing with a pressurized cabin, one must consider the effects of a sudden loss of pressure due to mechanical failure of the system, especially in the case of perforation of the cabin walls, battle damage, or loss of the bubble

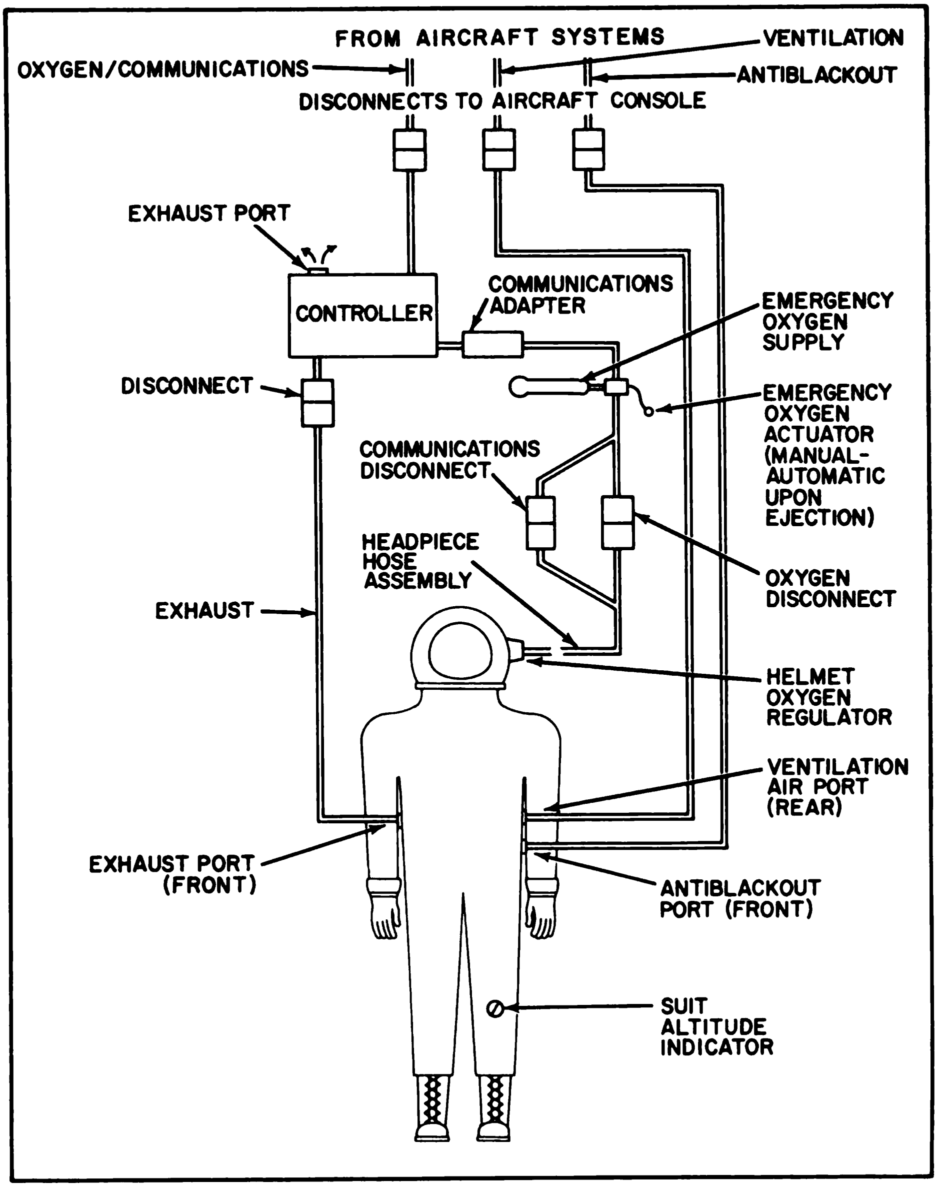

FIGURE 11–22. *Pressure suit system schematic* (NAVWEPS 13–10–501).

canopy. In the event of such a rapid decompression, the following factors will govern the decompression rate and the physiologic effects:

Cabin Volume: Other factors remaining constant, a larger space will take longer to decompress than a smaller one.

Size of the Opening: The relationship between cross-sectional area of perforation and the cabin volume will be the main factor determining decompression rate. The larger the perforation, the more rapid the decompression rate.

Pressure Ratio: (P cabin/P ambient): The larger the pressure ratio, the longer the decompression time.

Pressure Differential: The difference between ambient pressure and cabin pressure influences the severity of the decompression but not the rate. The larger the pressure change, the more severe the decompression.

Flight Altitude: Apart from the rapid decompression itself, the flight altitude determines the severity of the physiological effects on the body after equilibrium has been reached, particularly with reference to hypoxia and aeroembolism.

From the rapid decompressions which have actually occurred in flight it appears that the primary danger from the actual decompression comes from the possibility of being blown through the opening or struck by flying debris. There is some possibility of trauma from the sudden expansion of gas within the body but only if the glottis is fixed at the time of decompression, which is unlikely.

A method has been developed which may be useful in deciding the safety of pressure systems relating to possible decompression. Two formulas are used:

$$\text{RGE (theoretical)} = \frac{\text{Pc} - 0.91}{\text{Pa} - 0.91}$$

$$\text{RGE (maximum)} = 2.1 + \frac{3.79\,\text{Vc}}{\text{A}} \quad \frac{\text{Pc} - \text{Pa}}{\text{Pa}}$$

RGE = relative gas expansion

A = total cross-sectional area (square inches) of opening (largest plexiglass opening is suggested for calculations)

Vc = cabin volume (cubic feet)

Pa = atmospheric pressure (psi)

Pc = cabin pressure (psi)

If RGE (theoretical) is greater than RGE (maximum), danger is present. If RGE (maximum) is greater, the operating conditions may be considered safe.

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CHAPTER 12

Environmental Factors in Survival, Work, Injury, and Disease

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General
Thermal Equilibrium
  Heat Loss
  Heat Regulation in Cold Environments
  Heat Regulation in Warm Environments
Hypothermia
  General Hypothermia
  Local Hypothermia
  Special Problems of Operations in Snow
Hyperthermia
  Adverse Effects
  Acclimatization
  Heat Protection
The Problem of Water
  Normal Operations
  Sea Survival
  Land Survival
  Detection and Rescue
Training Films
References

GENERAL

SURVIVAL presents the problem of the extreme situation in which aircrew may find themselves as a result of an inflight emergency and concerns those actions which are necessary to remain alive until rescued in various hostile environments.

On the other hand, there are the problems of maintaining the functional effectiveness of personnel who must live and work under similar adverse environmental conditions. The Flight Surgeon should understand these interrelationships, because in many cases the major environmental stresses which an aircrew must undergo are those exerted by the preflight and post-flight environment. Furthermore, the acclimatized aviator is the one with whose health and function the Flight Surgeon will be dealing.

Man's response to extremes of environment has long been of interest. It had been realized even before commencement of tropical and polar operations that an individual's ability to lead a healthy and productive life in such environments was dependent on his successful adjustment to those physiological and psychological stresses peculiar to such environments. Although it is nearly impossible to divorce completely the physiological effect of temperature extremes from their psychological effect because of the great influence each exercises on the other, this chapter will consider primarily the former.

Because all mammals are homeothermic organisms, maintenance of the internal body or "core" temperature within a minimal range of variation, independent of ambient temperature, is the major factor responsible for the normal pattern of biochemical reactions in man. The biochemical reactions responsible for metabolism impose several requirements upon the mechanisms regulating body temperature. Since most biochemical reactions are effected through action of enzymes which are particularly sensitive to temperature change, a fall or rise of only a few degrees of core temperature may so retard metabolism that normal behavior is impossible and death may ensue. In the course of biochemical oxidations, heat is liberated, and unless provision is made for its dissipation, overheating of the body may occur which could cause death as a result of irreversible damage to the various enzymatic systems and cells of the central nervous system. It is mandatory therefore, that thermal equilibrium of the body be maintained.

THERMAL EQUILIBRIUM

Thermal equilibrium is preserved by the body's ability as a whole to alter its rate of heat production and heat loss. Since body temperature is really a measure of heat content or storage, a fall in temperature indicates a decrease, while a rise denotes an increase in the total heat content of the body. Although the core temperature varies normally over a range of only a single degree, the variation in temperature of the exposed portions of the body reflects its continual effort to achieve equilibrium as first the rate of heat production and then the rate of loss is altered by environmental and/or physiological changes. A normal sized, unclothed man at rest in a postabsorptive state can, with minimum body effort, reach thermal equilibrium at a room temperature of 86° F (30° C). Under such conditions the individual retains only that amount of heat formed by his basic metabolic processes which comprises normal storage, and loses the remainder to the environment without utilization of any of his reserve mechanisms of heat loss. The preceding example is only one instance of countless states of thermal equilibrium. Its significance lies in the fact that no reserve mechanisms of heat production or heat loss are required to achieve equilibrium. Under different conditions of clothing, activity, or environment, additional physiological adjustments would be needed to maintain an equivalent state.

Another expression of thermal equilibrium has been designated as the comfort zone. A gross definition of the comfort zone is those environmental conditions which cause neither sweating nor shivering. A more precise specification is possible through reference to the Effective Temperature Scale, which has become an accepted index of environmental comfort.

A Scale of Effective Temperature may be presented (see figure 12–1) as a family of curves formed by a plot of all combinations of relative humidity and temperature that yield the same subjective *sensation* of temperature. It is generally agreed that the optimum comfort range for persons wearing normal indoor clothing is +65° to +73° ET. As can be seen, this corresponds roughly to a temperature range between +70° and 80° F with relative humidity between 40 and 60 percent. Although in practice the Effective Temperature Scale has been found workable, it should be noted that the stated comfort range will be altered as a function of any variation in amount of clothing worn and level of physical activity, and with the introduction of the factor of air movement.

Heat Loss

Heat is lost from the body by several different physical processes: radiation, conduction and convection, and the vaporization of water.

Radiation is the transfer of heat from the surface of one object to another without physical contact between the two. The magnitude of heat loss in

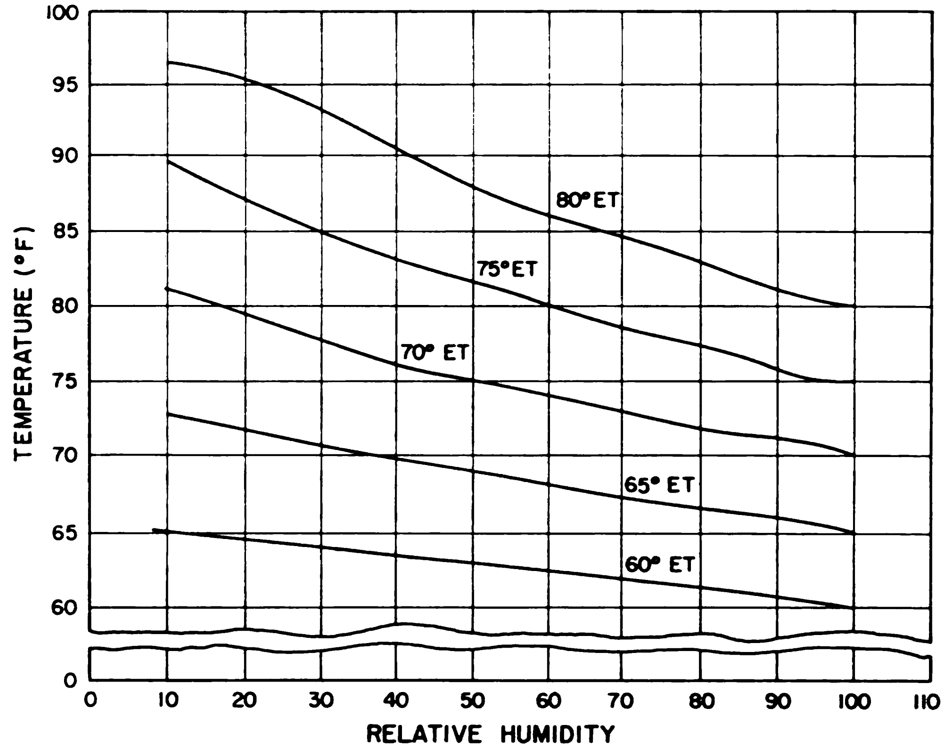


Figure 12–1. *Scale of effective temperature.*

man is directly dependent on skin surface area and the average temperature gradient between the skin and surrounding objects. The heat loss from radiation varies widely with environmental conditions. In a temperate climate, a resting individual, wearing ordinary clothes, loses about 60 percent of his heat production by radiation. At temperatures of 90° F, this loss may drop to zero. Conversely, at subzero temperatures, heat loss by radiation may reach levels higher than 60 percent.

Conduction and *convection* are less important methods of heat loss in temperate climates but assume major roles in polar climates. By conduction the cold air in immediate contact with the skin is warmed; the heated molecules move away, and cooler ones approach to take their places; these in turn are warmed and the process perpetuates itself. The air movements constitute convection currents. Any process, such as wind, which tends to increase the rate of movement of the ambient air relative to skin surface intensifies heat loss. This phenomenon has been incorporated into the concept of windchill by Siple (figure 12–2). A unit of windchill is defined as the amount of heat that would be lost in an hour from a square meter of exposed skin surface which has a normal temperature of 91.4° F. Given a hypothetical situation wherein the wind velocity is 20 miles per hour and the temperature is 34° F, reference to the Windchill Chart discloses that at the given wind and temperature conditions of the hypothetical situation, the rate of cooling of all exposed flesh is the same as at minus 38° F with no wind. It is easily concluded that under the climatic conditions observed in polar operations, conduction and convection are significant causes of heat loss and potential contributory factors in the causation of cold injuries.

Heat loss by conduction also occurs from transfer of heat to tidal air as it is warmed in the respiratory passages and lungs, water and foodstuffs taken into the gastrointestinal tract, and waste materials (urine and feces) as they are eliminated.

Vaporization of water removes heat from the skin surface and the moist mucous membranes of the respiratory epithelium because when one gram of water is converted into water vapor, 0.58 calories of heat must be supplied from the surroundings for the conversion to occur. Although the actual amount of heat loss depends on the ambient relative humidity, in Antarctica, where humidity is very low, respiration alone may account for 10 percent (375 calories) of an individual's total daily heat loss. Insensible perspiration, as is shown in a later section, accounts for an additional loss of about 400 calories.

Loss of heat by vaporization of perspiration from eccrine sweat glands may account for a large part of the total heat loss at temperatures of 93° to 95° F, or above, but in polar climates it assumes importance only under certain clothing conditions which will be discussed later.

It can be seen, then, that loss of heat from the body occurs primarily at two surfaces, the skin and the epithelium of the respiratory system; and under constant environmental conditions, the amount of heat loss depends upon surface area, temperature gradient, humidity, and thus vapor pressure gradient, and the rate of airflow over the surface.

Heat Regulation in Cold Environments

Some of the physiological methods used to diminish heat loss observed in lower mammals are unsuitable or impractical for man. Such procedures as rolling up into a ball and thereby markedly decreasing the area of exposed

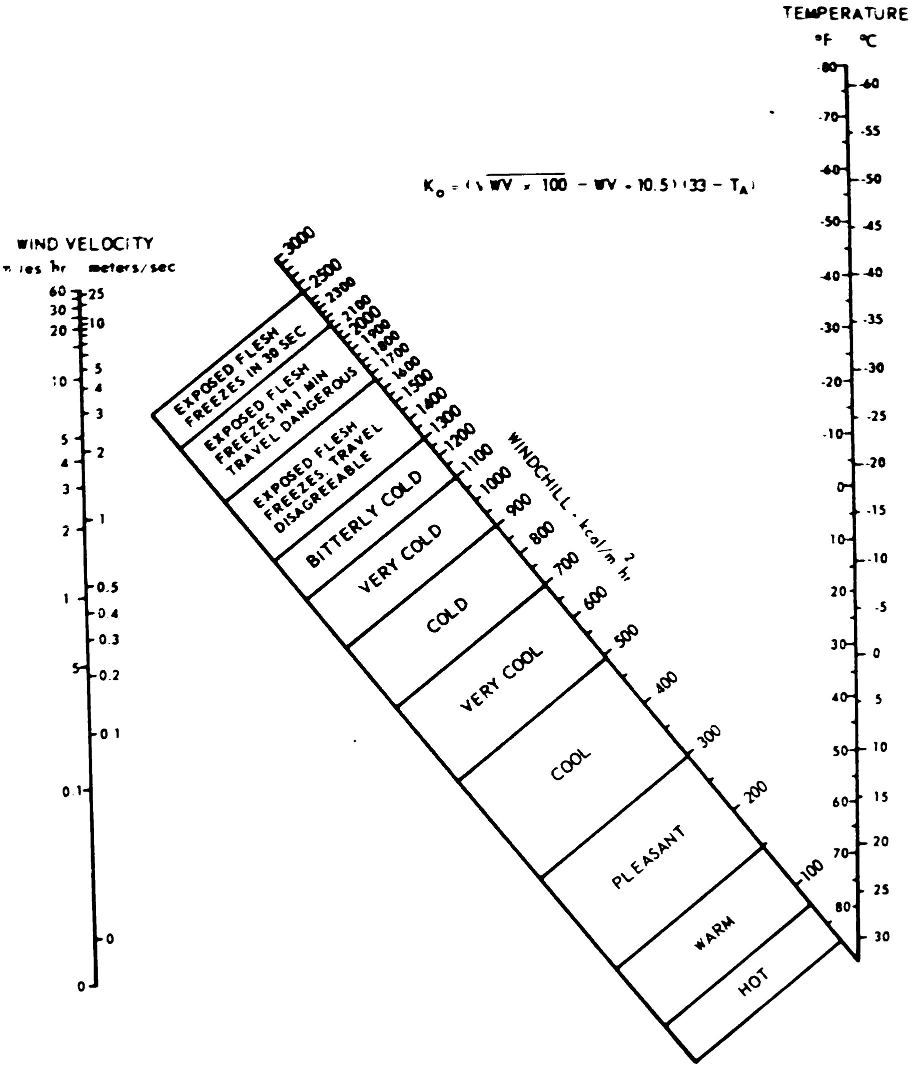


Figure 12–2. *The windchill index does not account for physiological adaptations or adjustments and should not be used in a rigorous manner. It is based on field measurements by Paul Siple during World War II* (blockley, 1964; source: consolazio et al.).

skin, or diminishing heat loss from vaporization of water at respiratory surfaces by cessation of panting, obviously have little application to human beings.

Since approximately 80 to 85 percent of heat loss occurs from the body surface, any reduction in skin temperature should conserve body heat.

Changes in the temperature of body surfaces are mediated through the activity of three physiological mechanisms. Unfortunately, one of the three mechanisms, although of some value in retaining body heat, is of greater importance when heat dissipation is desired.

The first mechanism depends upon the means by which heat is transported from the depths to the surface of the body. Blood is 80 percent water by volume; and, because of the water's high heat capacity, circulating blood is the primary source of heat transfer to the body surface. The total amount of heat brought to a given area is a function of the rate of blood flow through the area. If the rate of flow is retarded by local constriction of the superficial arterioles, the total heat transfer from blood to skin tends to be small and the skin remains cool, thereby decreasing the temperature gradient between it and the surrounding cold air. Although this physiological mechanism is an important means of heat conservation, a certain minimum blood flow must be maintained through the skin to prevent localized anoxia and cellular death.

The second mechanism is dependent on the phenomenon of horripilation, or erection of body hair, which increases the thickness of the layer of nonconducting air entrapped between the hairs. Since surface temperature depends upon the ease with which heat is transferred from the body to the environment, horripilation affords a satisfactory method for retarding conduction by reducing the temperature gradient between the skin and environment even though the skin temperature may remain at a high level. Due to evolutionary processes, man is poorly equipped to take advantage of horripilation. Nevertheless he utilizes the principle by substituting a different insulating material for the hair he lacks in the form of clothing. A single layer of clothing limits heat exchange by replacing the single temperature gradient between a nude subject and his environment with three such gradients. One of these three exists between the skin and the inner surface of the insulation, another exists between the outer surface of the insulation and the environment, and the third gradient is found between the inner and outer surfaces of the insulation. The effectiveness of clothing in decreasing heat loss is proportional to the magnitude of this third gradient, which in turn depends upon the nature and thickness of the nonconducting substance.

A third mechanism modifies the temperature of body surfaces by varying the amount of moisture available for vaporization. Since insensible heat loss due to perspiration and respiration is not subject to wide variation in cold climates, the primary value of this mechanism is in the dissipation of body heat rather than its retention.

When heat loss exceeds heat production in spite of utilization of the previously discussed physiologic mechanisms, the body, in an effort to regain thermal equilibrium, increases heat production. Heat production is essentially chemical in nature and is developed from at least two different sources. When body temperature decreases in a resting human exposed to cold, involuntary muscular contractions (shivering) ensue. Since only 25 percent of the energy liberated by chemical changes in contracting muscle is converted to work, heat production is equivalent to three or four times that of the muscle at rest. Even more efficient in heat production than isotonic involuntary exercise is voluntary isometric exercise (contraction of both extensors and flexors simultaneously) which converts all of the energy produced to heat.

Another source of heat production which does not involve skeletal muscle contractions has been demonstrated by various investigators in both animals and man. Small experimental animals like the rat are able to vary their rate of heat production within several hours by some mechanism in which the hormones of the thyroid gland, the adrenal cortex and, possibly the adrenal medulla participate without any detectable change in either voluntary or involuntary muscular activity. Both the adrenal cortex and thyroid undergo changes which can be detected histologically and by chemical analysis. A similar nonshivering-induced increase in heat production has been demonstrated in man by several investigators. At this time, insufficient information exists to describe the mechanism involved in man.

Those regulatory responses discussed above can succeed in maintaining thermal balance for hours within the compensable zone. Figure 12–3 shows the approximate limits of the compensable zones on both sides of the comfort zone. When temperatures fall below the compensable zone for cold tolerance, thermoregulation fails and extraneous methods must be utilized to maintain thermal balance.

Heat Regulation in Warm Environments

Physiological Mechanisms.—As the body becomes heated, whether it be from exposure to high temperature environment, physiologic exercise, or a combination of both, superficial vasodilitation occurs, thereby increasing the blood flow to the skin. This allows additional heat to be lost primarily by convection and radiation. Additional heat is lost through insensible perspira-

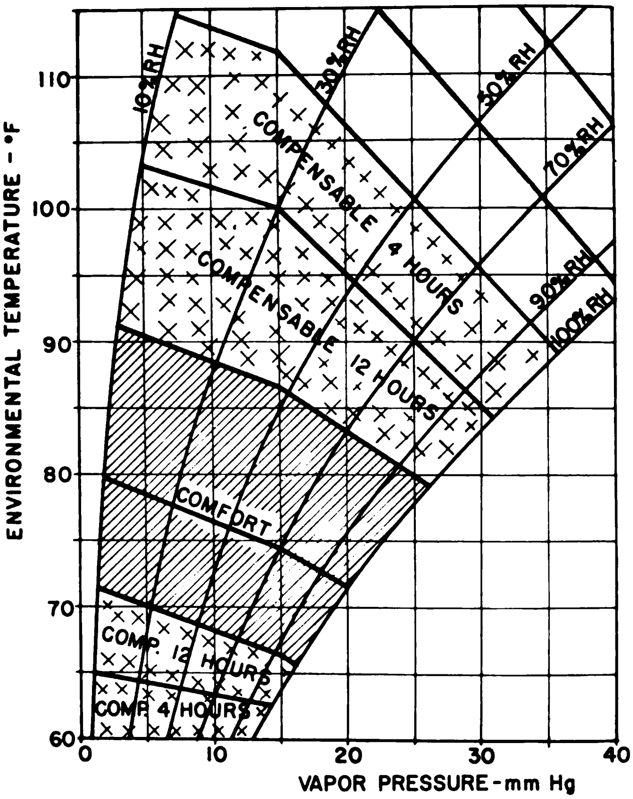


FIGURE 12-3. *Representation of comfort zone and compensable zones* (WEBB, 1961).

tion and respiration. When the body becomes heated beyond the limits of the comfort range, these processes are insufficient to maintain thermal equilibrium.

A second response to heat exposure is an increased rate of excretion by the eccrine glands. Through the evaporation of water produced by sweating, a large increase in the rate of heat loss can be realized. Although the body can produce sweat at a high rate, evaporative heat loss is limited by the physical process of evaporation which, in turn, depends upon skin temperature, water vapor pressure gradient, and movement of the air surrounding the individual. The vapor pressure gradient is defined as the difference between the water vapor pressure at the skin and in the surrounding air.

Effects of Clothing.—Thermal comfort and stability are modified considerably by the clothing an individual wears. With suitable clothing a person can achieve a level of comfort and survival over a much greater range of environmental temperatures than those shown in figure 12-3.

Heat transfer through clothing is a function of the thermal resistance of the clothing and the temperature and humidity differential between the inner and outer surfaces. Thermal resistance of clothing is expressed in terms of "clo" units. One clo is defined as the equivalent to normal indoor clothing and is that clothing insulation required to keep a resting/sitting man with a metabolism of 50-Kcal/m^{2}hr indefinitely comfortable in an environment of 21° C with an air movement of 20 ft/min., and with a relative humidity of less than 50 percent (Kerslake, 1965). The insulation value of clothing is a function of the air trapped between its fibers and is roughly equal to four clo per inch thickness of fabric.

The biophysics of clothing has become singularly significant in recent years because it is an interdisciplinary approach (physiology, psychology, physics, clothing design, and textile science) which relates human work efficiency and comfort to a specific task in a particular environment. Prior to this realization, the physiologist was consulted for information about the man and the climatologist was sought out for knowledge of the environment. Little was known about the physics of how clothing materials interacted with each other or with the complex man-clothing-environment system. Most experimental measurements utilized "steady state" conditions for simplicity and the reduction of variables. Utilization of the biophysical approach should result in an end product clothing system in which man, environment, and clothing are united into a functional and comfortable whole.

In a cold environment, an individual frequently finds himself wearing more insulation than he needs during work and less than he needs at rest. This is readily explained by considering the biophysics of the situation. For maintenance of thermal equilibrium in any given environment, the optimum insulation is five to six times as much at rest as at work. The problem, then, is to design clothing which optimizes the balance between static and dynamic insulating efficiencies. Specifically, the rate of heat loss must be minimized when activity level is low and increased when activity level rises.

Primary functions of protective clothing are to insure adequate ventilation for the escape of both insensible and sensible perspiration, and to provide around the body an insulating zone of dead airspace which is compartmentalized in sufficiently small pockets so that currents of air will not be set up by movements of the body and thus disperse heat.

Polar explorers have cautioned repeatedly against the danger of sweating profusely, because during later periods of diminished activity, excessive heat loss occurs when the vaporized perspiration condenses on the cold outer cloth, thereby permitting direct heat transfer by conduction. Because retention of vaporized perspiration in clothing diminishes the effectiveness of the sweat mechanism in cooling the skin surface, increased production of perspiration ensues and a potentially dangerous situation develops.

Conversely, in hot environments clothes become a barrier to the evaporation of perspiration from the skin because sweat evaporated from wet clothing is much less effective in removing heat from the body than moisture evaporated directly from the skin.

Figure 12–4 illustrates the interaction of clothing insulation, activity, and ambient temperature on the maintenance of thermal balance.

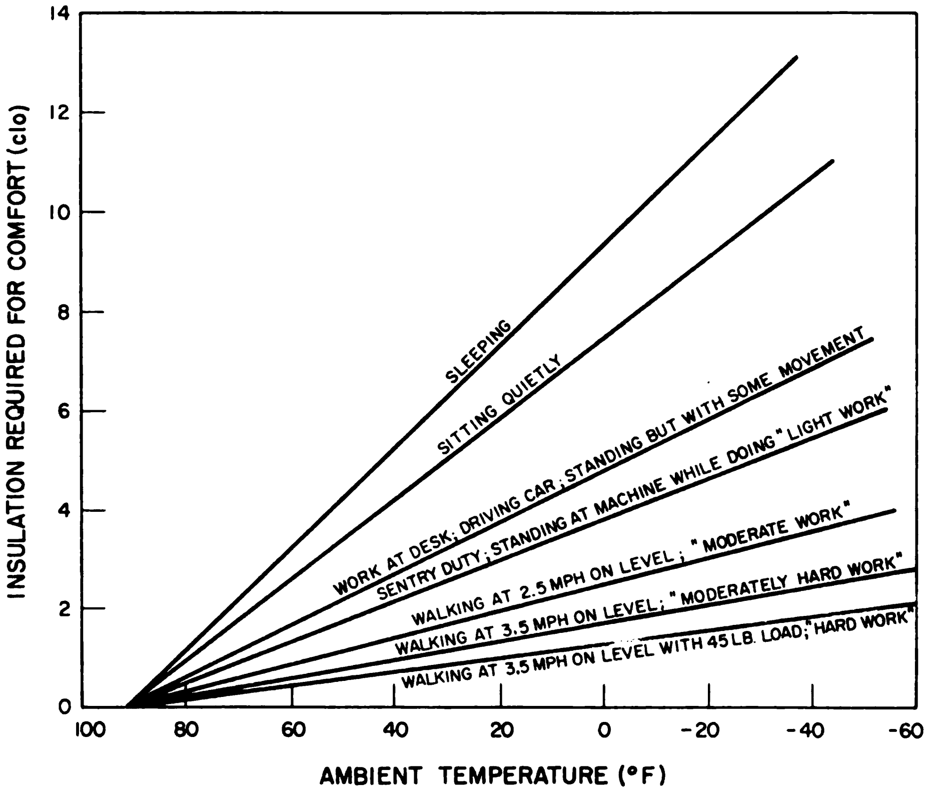


FIGURE 12–4. *Prediction of total insulation required for prolonged comfort at various activities in the shade as a function of ambient temperature. M is heat production in cal/m²/hr.* (FROM Human Engineering Guide to Equipment Design, C. T. MORGAN, J. S. COOK III, A. CHAPANIS, & M. W. LUND (EDS.), COPYRIGHT 1963, MC GRAW-HILL. USED BY PERMISSION OF MC GRAW-HILL BOOK COMPANY.)

HYPOTHERMIA

When heat loss exceeds heat production, hypothermic injury may develop. Hypothermia is classified as general or local, depending on whether the injury affects the individual as a whole or affects only a particular part of the body.

General Hypothermia

General hypothermia may result from total or partial immersion or from exposure to cold air alone and is the most frequent cold injury seen in naval aviation medical practice afloat. Deck crews and shore station crews must work outside in very cold weather but are usually prepared and equipped to resist the deleterious effects of exposure to cold air. The aviator landing in cold water with or without an exposure suit is an entirely different matter as is the shipwreck survivor and the man overboard. Water, because of its heat conductivity, has a cooling power 23 times that of air. Immersion in water at 28° to 35° F may result in unconsciousness in five to seven minutes and in death in ten to twenty minutes. The ungloved hand is useless in one to five minutes. In this critical situation, actuation of controls such as the inflation lever of the liferaft or the actuating handles of the lifevest or the parachute harness fittings must be accomplished immediately upon hitting the water. As soon as possible, thereafter, any other survival actions that are possible must be taken. Boarding of the inflated liferaft and donning of the survival poncho or inflation of the liferaft hood (if so equipped) must be done while finger dexterity remains. Any means available of insulating the skin or body against contact with the cold water should be utilized. Nevertheless, it should be noted that during World War II only one man in ten survived after one-half hour of immersion in 29° F water.

Other means of mere survival in such cold water are, of course, of interest. The means occasionally advocated by those unfamiliar with the physiological reaction is to commence violent exercise immediately upon entering the water. "In a few instances, men have saved themselves by violent exertion as soon as they hit the water, were able to swim a short distance, and pull themselves on the ice or up a ladder. More have died or would have without help because of muscle spasm. In water at 41° F for twelve minutes, it has been found that moderate work doubled the rate at which rectal temperature fell because of increased blood circulation. Working as hard as possible only slightly decreased the rate of temperature loss at this temperature. If in a marine disaster at temperatures not causing the above acute reaction, exert oneself as little as possible in order to live longer.

"Any clothing worn will of course reduce the heat transfer between the body and the water and, thereby, increase the survival time." (Hedblom, 1965)

Once core temperature has dropped to 95° F, thermal control is lost and the body is no longer homeothermic. Coma occurs and death ensues when core temperatures reach 79° F.

Subjects in Rascher's experiments at Dachau placed nude in water from 35 to 53° F experienced severe pain, which gradually disappeared, in the first five to ten minutes. There was a chill later associated with clonic or tonic convulsions. Speech became difficult, and inhibition of breathing was associated with muscular rigidity. Respiration was at first rapid and then dropped to 24 per minute. Complexion was first pale and then cyanotic. Pulse rose at first to 120 beats per minutes, but slowed at rectal temperatures of 94° F and below until it reached a rate of 50 per minute. Total irregularity of the heart occurred at rectal temperatures of 85° F. Carried to fatal termination, cardiac arrest followed the cardiac arrhythmia.

In this discussion, some of the factors influencing survival in cold water have been mentioned. Figure 12-5 outlines the voluntary tolerance wearing flight clothing for exposure of human subjects in numerous experimental studies, including a recent one using a diver's wet suit in conjunction with a flight suit and long underwear. Such experiments are typically terminated when the subject declines to accept the discomfort any longer or reaches a skin temperature below 50° F. The second limit shown, pertaining to men protected by potentially waterproof garments, reflects the fact that hands and feet cannot be adequately insulated and remain functional. The third curve noted in the figure shows the longest survival durations noted while wearing antiexposure suits. The extent to which real survival time would exceed this limit is difficult to predict due to the importance of injury, equipment available, and such psychological factors as belief in the possibility of rescue (Blockley, 1964).

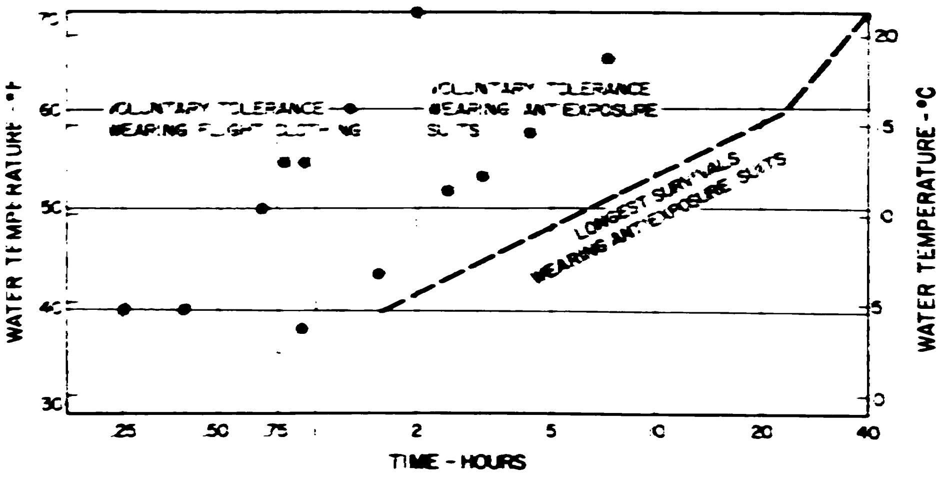


FIGURE 12–5. *Survival in cold water.* (BLOCKLEY, 1964; SOURCES: BECKMAN & REEVES; DAMATO & RADLIFF; HALL ET AL.; MC CANCE ET AL.; MOLNAR; & U.S. NAVY.)

An analysis of over 25,000 personnel on ships lost at sea during 1940–1944 showed that of those who reached liferafts, half died by the sixth day if the air temperature was below 41° F; survival time increased with increasing air temperature.

From this study (Molnar, 1956), the standard chart of Survival Time Versus Water Temperature in Degrees Fahrenheit as contained in NAVWEPS 00–80T–56 was drawn. This chart is now believed to be in error, since it indicates an almost unlimited survival in water at 68° F or above. However, Beckman and Reeves (1966), in an excellent controlled study, have shown that even with water temperature at a relatively high 75° F, many subjects could not complete a 12-hour immersion while nude but wearing the MK–2 lifevest. The factors associated with inability to complete the 12-hour period were:

1. *Decrease in core temperature,* representing a failure of body heat production to match the conductive loss. This occurred despite an increase of energy production of almost three times over the pre-immersion resting control value.

2. *Muscle cramps.* Over half the subjects were unable to complete the study because of severe, persistent, and disabling cramps in major muscle groups including quadriceps femoris or erector spinae muscle groups. These cramps usually began after three to four hours of immersion at 75° F and gradually worsened, and were relieved only by removal from the water. In

several cases, cramps were associated with a blood glucose level of less than 60 mg/100ml.

3. *Diuresis.* In the subjects who completed twelve hours of immersion, urine output increased over four times the control value in the first four hours, returning to control values at the end of the twelve hours. Concurrent hematological studies showed hemoconcentration.
4. *Changes in corticosteroid levels* indicated that immersion stress produced a significant adrenocortical stress response.

Therefore, exposure to extremely cold water during immersion produces paralysis and rapid physiological deterioration leading to early death by cardiac arrest. Exposure over much longer durations to considerably higher temperatures allows other physiological mechanisms to act, resulting in different causes for survival durations during immersion in 75° F water as compared to 29° F water. Nevertheless, a mechanism common to the entire spectrum of water temperatures as contained in the oceans over the world is a decrease in core temperature due to the mechanisms noted previously.

Since this is such an ever-present hazard to the naval aviator, protective devices have been designed and furnished based upon the principle of insulating the body from the sea. A primary device for doing so is the liferaft with its associated survivial equipment and materials. For use in cold climates, a continuous-wear antiexposure suit has been developed and furnished to all naval aviators for use as directed by NATOPS procedures. In addition, any clothing worn at the time of entry into the sea will act as an insulating barrier against heat loss to the sea and should be retained if possible. After boarding the liferaft, ordinary flight clothing will prevent development of cold air injury to the tissues, at least to some degree.

An excellent review of the known data concerning survival in sea immersion and cold injury due to immersion is Whittingham (1965).

Treatment of General (Immersion) Hypothermia

"With a large number of cases, as in shipwreck, treat those not breathing (but alive) and the unconscious first. Pouring water at 110–116° F over those waiting for treatment will increase the number of survivors, for after removal from the water (without treatment) a paradoxical 'afterdrop' in temperature, caused by vasodilation with rapid cooling of the core from the cold 'shell' tissues, will kill many who seem safe" (Hedblom, 1965).

Usually, the treatment for general hypothermia due to immersion is rapid rewarming. If the patient is conscious enough to move and without extremity injury or frostbite, he may be placed in a tub or shower at 108° F to 110° F and left there until he stops shivering. Most aircraft carriers have no tub available within which to conduct a rapid rewarming. An acceptable substitute which is generally available aboard ship is a multiperson liferaft, such as the eight- or twenty-person raft which can be inflated in an area in close proximity to a supply of hot water and filled with water at a temperature of 108° to 110° F. Such an improvised tub allows for the treatment of multiple casualties, while conserving the ship's fresh water supply. When the patient immersed in such a tub begins to sweat, the heat "debt" can be presumed to be reversed and a positive heat balance available in the individual. During rapid rewarming, hot coffee should be given, both to cause mild stimulation and to increase the heat input into the body. As soon as sweating begins, the individual can be moved from the tub and should then be warmly clothed and forced to ambulate. Ambulation increases the body's heat production and, in addition, tends to prevent aspiration and pneumonia. The major danger in rapid rewarming is that the rewarming cannot be carried out fast enough, in which case the patient may go into shock due to paradoxical cooling.

If the patient is unconscious but breathing, place him in a tub or shower with water at 108° to 110° F and keep him there until he begins to sweat, regardless of when he regains consciousness. Usually patients will regain consciousness long before they start to sweat. Thereafter, the patient may be treated as above with warm clothing and forced ambulation. An airway must be established early and maintained.

Management of those patients who are unconscious and not breathing is quite difficult. If the heat loss cannot be rapidly reversed, most supportive measures designed to maintain oxygenation of the blood and circulation of the blood, even in the presence of cardiac arrhythmias, will be of only limited effectiveness. Therefore, rapid rewarming in a tub with a water temperature *of 108° to 110° F* is a *necessity*. Simultaneously, however, oxygenation of the blood by mouth-to-mouth resuscitation or other means, and electrical and/or chemotherapeutic defibrillation must be accomplished.

Hedblom (1965) notes that fibrillation may continue for up to two hours after the body temperature has returned to normal. Any electrical defibrillation must be accomplished prior to commencement of rapid rewarming in a liquid medium, and care must be taken that no electrical accidents occur during the excitement and confusion of treatments of considerable numbers of patients simultaneously. If hypovolemic shock is present, it should be treated in the usual way with dextran or plasma.

German experience in World War II indicated that the earliest possible use of rapid rewarming was so important to survival of their aviators rescued from the English Channel that they even equipped the small motor torpedo boats utilized for rescue with the necessary hot water tubs. Statistical information on the success of these efforts is not available, but the point to be remembered is that patients removed from a cold water immersion event should be evacuated as quickly as possible to the nearest place where rapid rewarming can be accomplished, and conversely, rapid rewarming facilities must be made available as near as possible to the place of rescue.

Therapy of general (immersion) hypothermia must still be considered as open to considerable debate. NAVMED 5052–29 (1964) indicates that rapid rewarming is contraindicated in treatment of general hypothermia. However, the emphasis in that document is upon the patient who has become hypothermic ashore and not because of immersion. It is believed, as indicated previously, that *immersion* hypothermia *must* be treated by rapid rewarming, with the full knowledge that the treatment regime for the shipboard physician will be considerably different from that possible, and perhaps even desirable, in a large, modern hospital ashore.

When operations in cold climates are anticipated, the Flight Surgeon should prepare for the environmental hazards expected. He should thoroughly familiarize himself with prevention and treatment measures. Excellent, detailed materials (beyond the scope of this manual) are available. *The Polar Manual* (Hedblom, 1965); *Low Temperature Sanitation and Cold Weather Medicine,* NAVMED 5053, 1954; and *Cold Injury,* NAVMED 5052–29, 1964, should be obtained and reviewed prior to deployment.

Local Hypothermia

In his *Polar Manual* (1965) Hedblom gives the following discussions concerning local hypothermia:

There are two types of local hypothermia: frostbite, superficial or deep (freezing); and immersion or trench foot.

Superficial Frostbite.—In order to clarify the considerable confusion of terminology in the literature regarding frostbite, freezing, trench foot, etc., *frostbite* may be defined as a *skin* condition similar to sunburn, little deeper, and classified into two degrees of severity in the same manner: first degree (redness, followed by branny desquamation), and second degree (blister formation in 24 to 36 hours, followed by sheet desquamation). It is most common following brief exposures to extreme cold. Below 20° F, the ease of contraction depends directly on the windchill factor (relation of wind to temperature), duration of exposure, and the adequacy of protection. Accompanying this condition per se, there is rarely a remarkable lowering of the general body temperature.

It is common on the face, hands, and feet, being the most troublesome about the face. Its onset is signaled by a sudden blanching of the skin of nose, ear, or cheek, which may be subjectively noted by the experienced as a momentary tingling or "ping." Subjectively the face muscles won't work. Under severe conditions, the "Buddy System" of two men watching each other's faces for the telltale yellow-white spots will minimize tissue damage by early detection. In severe cold if your face, hands, or feet STOP hurting, *investigate*—you probably have frostbite. When palpated, unthawed frostbitten skin may be cold and frosty, crisp or resilient. It may move freely over bony prominences (joints, knuckles, facial bones). Thawed superficial frostbite may be indistinguishable from deeper freezing injury.

Figure 12–2 shows that human flesh freezes at cooling values of 1,300 to 1,500 calories per square meter per hour. With a wind velocity of 22 miles per hour, exposure at temperatures of 17.6° F for one hour, or minus 22° F for one minute will cause human flesh to freeze. This graphic demonstration emphasizes the importance of windchill as a causative agent in frostbite.

To avoid superficial frostbite of the face, a moderately stiff 4- to 5-inch tunnel extended in front of the facial aperture of the parka protects the face from wind at all but about 90 degrees dead ahead. This should contain a malleable wire at the outer aperture so that the opening may be shaped With wolverine or wolf fur circling this aperture, ice tends to accumulate from breathing. Frequent beating of the fur minimizes this difficulty.

Face masks are particularly needed by aircrews who must occasionally work in the slipstream of aircraft tuning up on the ground. A windproof pile-lined band, which will cover cheeks and tip of the nose only, is satisfactory under many conditions.

Windproof leather gloves or mittens are most satisfactory for protecting hands. Particular care should be taken not to let the hands get wet with kerosene, gasoline, alcohol, or other fluids which freeze below 32° F, for these will cause quick frostbite and freezing. Touching of very cold, bare metal with warm, moist, bare hands results in the skin sticking to the metal, with resultant loss of tissue or quick frostbite.

Foot gear MUST be roomy enough to permit easy movement of the toes, for continuous flexion and extension of the toes increase blood circulation and delay frostbite and freezing, particularly in the case of immobility of concomitant injury suffered in trail or plane operations. To be *particularly avoided* are flying boots or galoshes which are worn over ordinary shoes. Frozen feet are the result of wearing these, because the inner laced shoe does not permit swelling concomitant with frostbite or shock.

Clinically it has been proved that the quick thaw of *freezing injuries* in a water bath of 40° to 43° C (104° to 108° F) has a specific benefit on ultimate tissue recovery. Such injuries should be treated immediately whenever encountered, lest they progress to a freezing injury.

In the field, frostbite of the face is thawed by placing a warm hand over the spot until it hurts again. Frostbite of the fingers is best treated by wearing a parka with sufficiently large armholes that the arm may be withdrawn within the parka sleeve and the hand warmed under the opposing armpit. Thawing in this manner through the open front of a conventional coat allows rapid loss of vital body core temperature. Frostbite of the feet is best thawed on the warm belly under the parka of a trailmate. Warming of the heels, as well as numb toes of frostbitten feet permits quicker thaw and less danger to the toes.

Petrol pocket warmers inside big survival mittens or parka pocket assure that the subject will always have a warm hand with which to thaw out face spots. Under NO conditions should frostbite be treated by rubbing, with or without snow or slush. When frostbite begins to peel, as does sunburn, any bland lanolin-base ointment will allay discomfort. (Though *frozen* tissues swell and blister, resembling frostbite or burns, they are NOT to be treated with ointment as are burns.)

Deep Frostbite (*Freezing*).—When ice crystals form in tissues deep to the skin and its immediate subcutaneous tissues, an extremity is *frozen*. This is the third- and fourth-degree "frostbite" of literature. As "degrees" cannot be clinically distinguished before or during treatment, and as treatment is the same for all deep frostbite regardless of "degree" or duration, why quibble about degree? Freezing is always preceded by frostbite. It occurs most critically in the feet, occasionally in the hands and ears.

Unthawed it is painless, and tissues have a pallid yellowish color and appear somewhat translucent or waxy. Skin will not roll over bony prominences. Members may become quite solid or "wooden" to palpation, but never brittle. When brought indoors, the skin will collect droplets of moisture from the atmosphere just as a cocktail glass "sweats." Without rapid rewarming, blisters appear in 12 to 36 hours. Red-violet discoloration appears spontaneously on the first to fifth day. (This is not the slate-gray discoloration observed distal to arterial occlusion.) Termination without proper treatment is usually dry gangrene. Residual hypesthesias, paresthesias, and sensitivity to cold in recovered extremities are probably due to anoxic injury to the nerves from long, wet, cold exposure (trench foot) suffered prior to actual freezing. These seem to be more severe in frozen extremities which have not been rapidly rewarmed.

To prevent freezing:

1. Prevent frostbite (above).

2. Maintain "core" or general body temperature with adequate clothing, nutrition, hot meals, and hot fluids.

3. Avoid excesses of ALCOHOL in potentially freezing situations. It promotes excessive cooling from peripheral vasodilation (the subjective "warm" feeling), but more important, its narcotic sleepiness and euphoria, causing loss of the judgment, perception, and ambition necessary to fight cold, contribute to freezing injuries and death from general hypothermia.

4. Avoid excessive fatigue, mental or physical, for, like alcohol, fatigue causes neglect as well as lack of strength for proper preventive measures.

In the field, the following first aid measures should be observed in the treatment of freezing injuries:

1. Rewarm patient's *core* temperature. (See *Hypothermia.*)

2. There must be NO constriction to circulation from boots above or over the frozen area. Immobilize concomitant fractures loosely without traction, for the snug bandages necessary for traction will further jeopardize circulation and increase freezing damage. For the same reason, avoid pneumatic splints.

3. Never THAW or rewarm a FROZEN extremity until arrival at a medical facility with water, heat, power, and equipment for sterile bed care where extremities can be RAPIDLY rewarmed, for the following reasons:

a. *Medical*

(1) If thawed and REFROZEN, loss of digits and perhaps a hand

or foot is the invariable outcome from gangrene, which occurs in four to seven days.

(2) Rapid rewarming is a specific therapy which minimizes ultimate tissue loss and sequellae. Rather than thaw in the field, it is preferable to keep an extremity frozen for four to eight hours more to enable rapid rewarming and immediate hospital care.

(3) The swollen, edematous, painful, thawed extermity is more subject to infection during transportation than is the frozen extremity, and INFECTION is the chief reason for loss of tissue.

b. *Logistic*

(1) A man with a frozen extremity is NOT a stretcher case unless he suffers concomitant fracture or other serious injury. Under survival conditions, Freuchen walked miles and days for help, keeping his leg frozen, knowing that should it thaw he would be helpless.

(2) A stretcher case requires at least two men to carry him or pull him on an akja or toboggan.

(3) An ambulance, truck, or helicopter will haul only 4 to 6 stretcher cases, but they will haul 6 to 24 men, with one or more frozen extremities each.

4. *NO ALCOHOL,* vasodilating drugs, anticoagulants or friction.

When hospital care is available:

1. Rewarm patient's *core* temperature. (See *Hypothermia.*)

2. Routinely and vigorously treat shock with elevation of feet, warmth, oxygen and intravenous blood plasma or fluid should they be necessary. These patients, particularly those with concomitant injuries, often go into *profound shock* on admission to a hospital facility where it is warm.

3. If still frozen, *rapidly rewarm* in tub or water bath with pump or paddle, open-top washing machine, or whirlpool bath above body temperature but not "hot" to normal hand. Water at 102° F is "warm" to a normal hand; 116° F is as hot as the average person can stand continuously. A safe temperature seems to be midway between the two. Dr. Rudolph Campbell of Switzerland, Chairman, International Commission for Alpine Rescue, prefers increasing temperature from 50° F over 30 minutes to a final 102° to 106° F. Mills and his associates in Alaska have successfully used 105° to 112° F. NEVER go over 115° F or use dry heat, for this will superimpose a burn on the already insulted tissues. Thawing may take from 20 minutes

to an hour. It should be continued until all blueness or paleness of the digital tips has turned pink to burgundy-red and NO LONGER.

Man, suddenly thrust into an extremely cold environment under survival conditions, through fear (often stark terror) often becomes psychotic. When he observes his pale, painless, frozen extremity turn burgundy-colored with more or less painful rapid rewarming, more fear is generated. These patients may require protective measures, tranquilizers, or morphine for immediate thawing pain, and sometimes force is needed to bring the first thawing to the proper end.

4. If *already thawed,* DON'T rewarm.

5. Administer a tetanus booster upon admission.

6. Physiotherapy procedures are listed below:

a. Twice daily ½-hour whirlpool water bath (90° to 98° F), containing a mild detergent or soap until healing or spontaneous amputation takes place.

b. Immediate *ACTIVE JOINT MOTION,* particularly during hydrotherapy. This usually requires supervision and encouragement to overcome patient's pain, laziness, and/or apathy.

c. Buerger's exercises 20 to 30 minutes every four hours during day.

(1) Patient supine, legs elevated at 30° angle: 2 minutes.

(2) Patient sits on edge of bed, feet dangling:

(a) Flexes and extends ankle..........
(b) Rotates lower leg.................
(c) Spreads and closes toes............
} Slowly and deliberately 3 minutes.

(3) Patient flat in bed with legs under blanket: 5 minutes.

(4) Repeat above cycle three to six times per session.

Surgical care procedures follow:

1. For the first two to three weeks until the skin is dry and without blebs or lymph drainage, ALL attendants MUST use sterile isolation technique with masks, gowns, gloves, bedsheets, etc., to minimize infection. Treatment of elevated member under cradle is open without dressings, bandage, or ointment. Sterile cotton between toes minimizes maceration.

2. Blisters invariably complicate freezing injuries. *Early and clear blisters,* particularly down to the tips of digits, are a good sign. Higher blisters, and particularly blood-filled blisters are an ominous sign that perhaps a digit will be lost. *Leave all unbroken blebs alone.* For the first two to three weeks, debridement is limited strictly to trimming gross skin flaps loosened by the daily hydrotherapy. When the wound is dry and uninfected, constricting

digital eschars interfering with joint flexion may be slit laterally or dorsally. *Trust the whirlpool* to atraumatically and aseptically clean up local infection and to debride blisters and eschars.

3. NO AMPUTATION FOR AT LEAST THREE MONTHS or until after autogeneous (spontaneous) amputation, unless there is overwhelming infection or concurrent injury which requires it! This will minimize tissue loss and ultimately reduce hospital time by giving healthier tissue for such repairs or skin grafts as may be necessary. *Color of skin or amount of skin loss* below the line of demarkation is NOT indicative of end result. Skin can always be replaced; a prematurely amputated digit, hand, foot or leg CANNOT!

4. Split thickness or pedicle skin grafts when denuded granulating areas are ready.

5. Recovered dry frozen members are usually relatively painless, though they often have intrinsic muscle and fat pad atrophy. After six months, if disabling *wet cold* sequellae are improved by sympathetic block, sympathectomy should be performed.

Broad spectrum antibiotics are administered ONLY with evidence of deep infection or cellulitis.

In providing psychiatric treatment, opiates should be avoided after initial thawing because these cases are prone to addiction. During the first three months, pleasant environment, frequent visits, encouragement, and occupational therapy are mandatory. Depressed patients often talk surgeons into amputation because of depression over black foot or finger. Tranquilizers, alcohol, barbiturates and/or dextroamphetamine may be indicated during this period. (The Flight Surgeon often needs this therapy himself during this period, for his urge to do something surgical makes him the patient's and his own *worst enemy.*)

Ascorbic acid	50 mgm q4th
Hesperidine (or Rutin)	50 mgm q4th
Nicotinic acid	50 mgm q4th
Vitamin E (Alpha-tocopherol)	30 mgm daily
Vitamin A	25,000–50,000 units daily

High caloric, high protein diet.

The most promising new therapy for freezing injuries is IV administration within an hour after admission of 1½ gms/kgm body weight of ten percent low-molecular weight (41,000) dextran in normal saline, repeated BID for the first five days. This must be given *very* slowly to prevent overloading the heart. It is reported that in northern Sweden the LMWD dosage is routine 500 cc/day for the first eight days.

Smoking is discouraged.

NO anticoagulants, NO vasodilating drugs, NO high-intensity sound therapy.

Immersion Foot (*Trench Foot*).—Immersion foot results from wet exposure for hours or days of an extremity or portion thereof at temperatures *above* freezing. Dependency and/or immobility of the extremity aggravates and predisposes. Sailors in sea water or soldiers with wet feet in trench, rice paddy, or foxhole get the same condition. Nerve, muscle, and blood vessel injury due to cooling is the common feature. General body chilling and venous stasis are probable etiological factors (Hedblom, 1965).

Military operations in Vietnam have resulted in warm water immersion foot injuries (Anderson, 1967). In a single operation, 130 injuries from this one cause resulted. In these cases the injury consists of whitening and wrinkling of the skin and pain in the feet after two days or more of water exposure. Further exposure results in erythema on the weight-bearing surfaces, and edema. Complete recovery occurs following proper foot care.

In a seven- to ten-day military operation in which continuous wet foot exposure occurred, Anderson found that about 30 percent of the troops required evacuation for immersion foot injury. This could be prevented by drying the feet for six to eight hours per day with boots and socks off, but such is rarely possible in the military situation.

Enemy troops did not have this problem, since they usually were barefoot or used open sandals; however, South Vietnamese wearing U.S. boots and socks had the same immersion foot problems.

In Vietnam, Anderson found that silicone grease (manufactured by Dow Corning), 1 to 1½ ounces applied once daily, was quite effective in preventing immersion foot injury.

The entire area of diagnosis, treatment, prognosis, and prevention of immersion foot is described at considerable length in *Cold Injury,* NAVMED 5052-29 (1964), which should serve as the guide for the line officer as well as the medical officer in meeting this important problem. Hedblom's *Polar Manual* (1965) also has a great deal of material concerning immersion foot.

Special Problems of Operations in Snow

In his *Polar Manual* (1965), Hedblom indicates that operations in snow environments present two additional considerations which are unrelated to the maintenance of thermal balance but which can pose serious problems to both ground and flight personnel.

Snowblindness.—Snowblindness results from the burning of the conjunctiva by ultraviolet rays of 200 to 300 millimicrons wavelength and is contracted both when the sun is shining brightly on the snow and on overcast days. *There is no warning in snowblindness* since the symptoms are delayed from two to twelve hours. The symptoms are:

1. Severe pain due to the swelling of the conjunctiva.
2. Photophobia. The person is unable or reluctant to open his eyes because of the pain.
3. Lacrimation.
4. Smarting eyelids.
5. Headache and depression.

The only effective means for prevention of snowblindess is through use of dark glasses. Glasses should be of the side-blinder type, or goggles, to prevent eye damage from lateral light. To allow for both good vision and comfort from glare, light transmission should be approximately 15 percent. In extremely low temperatures, metal-framed glasses should be bound with a material such as plastic to prevent local frostbite of the cheek.

Such glasses must be worn out-of-doors at all times when the sun is above the horizon. The glasses are electronically coated with nickel on the front of the lens, with the coating heaviest at top and bottom and grading imperceptibly toward a clear area in the lens center. This double-gradient-density lens thus simulates the Eskimo slit goggle but permits a correction to be ground into the lens.

Snowblindness is best treated by:

1. Rest in a darkened room with bandaged eyes and cold compresses (for pain) for one to five days.
2. Butyn drops or ointment for extreme local pain. DO NOT USE COCAINE because of its softening effect on the corneal epithelium.
3. Sedatives, barbiturates, or tranquilizers, to counteract depression.

4. Atropine or homatropine drops for extreme or painful ciliary spasm (tiny, painful pupil of eye).

5. Antihistamines orally and cortisone drops may help.

Whiteout.—Whiteout is a daylight optical condition occurring on snowscapes with almost complete snowy obliteration by freshly fallen or blown snow, with the rays of the sun diffused by altostratus or cirrostratus clouds. Light is reflected back and forth from snow to clouds until everything is diffusely lighted with nonpolarizable light. The horizon disappears and there are no shadows. This condition can occur in perfectly clear ground conditions.

In the average whiteout, man on the ground without horizon ahead becomes disoriented and falls unless he can see another man, trail flag, or tractor ahead to discern which way is up. Crevasses, vehicle and man tracks, and snowy obstructions become invisible. Fliers in conventional aircraft must go on instruments, and a number of helicopter pilots, both Arctic and Antarctic, have become disoriented, flown into the snow, and been killed.

The problem posed by whiteout obviously is one of disorientation both for ground personnel and aircrew. It is fortunate, however, that complete whiteout is rare. There is usually some amount of horizon visible somewhere.

Some type of glasses must be worn to attenuate the effects of whiteout. Although red glasses are good, they have many well-known limitations for flight personnel. Yellow glasses are also good but the high light transmission through most yellow lenses allows glare to remain as an important problem. Gradient density neutral-gray glasses are minimally beneficial. Yellow lenses *with nickel coating* increase visibility remarkably during whiteout.

Glasses worn by fliers when in control of aircraft on previous polar expeditions, which have given good results, are the Bausch and Lomb Kalichrome C yellow Lenses, with Uniform plus *Top* Gradient Density "Inconel" metallic coating (the bottom density is omitted to make it easier to see instruments) in the "Outdoorsman" frame.

HYPERTHERMIA

Heat has always been a factor to be reckoned with in aerospace medicine. Whether concerned about the summertime heat in the cockpit from the exhaust manifold of early aircraft or the heat overload suffered by astronauts during EVA on the space flights late in the Gemini series, the problem is basically that of adapting man to the heat load by training, acclimatization, or equipment.

NAVMED 5052–5 (1957) concerns "The Etiology, Prevention, Diagnosis, and Treatment of Adverse Effects of Heat." Any Flight Surgeon whose unit is operating in a warm environment should have a copy of this publication readily available.

Speaking somewhat generally, when personnel are exposed to hot climates, the maintenance of thermal balance, comfort, and efficiency depends primarily on the extent to which heat dissipated by evaporation can offset the heat gained from convection, conduction, radiation, and metabolic production. In desert environments, where humidity is relatively low, heat is gained by convection from the hot ambient air; conduction from the ground; radiation from the sun and ground; and metabolic heat production. In the tropic jungle, discomfort for a given dry bulk temperature is even greater because of the higher humidity and low rate of air movement. The vapor pressure gradient across the skin is lower when the humidity is high, and thus the evaporation rate of sweat is decreased, although the sweat output will be markedly higher.

Adverse Effects

The adverse effects of hyperthermia have been subject to wide variations in classification. For the sake of simplicity, the disease states of general hyperthermic etiology will be limited to three syndromes. These are well covered in terms of diagnosis and therapy in NAVMED 5052–5. They are: ***heat cramps, water and/or salt-depletion heat exhaustion, and heat stroke or heat hyperpyrexia.***

Heat cramps are severe cramps of voluntary muscles which may occur during heat exposure due to salt depletion. These cramps are easily relieved by replacement of sodium chloride by mouth or intravenously.

In ***heat exhaustion,*** both salt-depletion and water-depletion types of etiology of heat exhaustion may occur, but the water-depletion type is quite rare and may be disregarded in ordinary practice. Therefore, the salt-depletion type of heat exhaustion is the condition with which the Flight Surgeon should be most familiar.

The syndrome is characterized by profuse perspiration, skin pallor, hypotension, and other manifestations of peripheral circulatory collapse. Other symptoms include headache, mental confusion, vertigo, incoordination, drowsiness, anorexia, weakness, and visual disturbances. Consciousness is rarely lost but the pulse rate is elevated (140–200). Principal laboratory findings include hyponatremia and a virtual absence of urinary chloride and sodium.

The mortality rate is quite low and as a rule, the removal of the patient to a cool environment, rest, and the administration of saline solution, orally or intravenously, will result in fairly prompt recovery, but treatment in bed should be continued until urine chloride (as NaCl) exceeds 3 gm/liter, which may take several days.

This condition is encountered fairly frequently in tropical flying, especially when long periods must be spent awaiting takeoff, with inadequate cockpit ventilation. Pilots should be warned that if they begin to feel extremely weak, or seem to be sweating profusely, along with dizziness, vertigo, or headache, the flight should be canceled. Even if takeoff is safely accomplished, the salt depletion can only become worse with the passage of time, and the probabilities of an inflight emergency are increased.

Prevention consists of increasing salt intake at mealtime, especially in the unacclimatized aviator, and maintaining a state of awareness of the condition in the aviator population.

Heat stroke is a very serious condition with a high mortality rate. Because of its seriousness, most physicians are familiar with its clinical features and proper therapy. Reference again should be made to NAVMED 5052–5, where the subject is well covered.

Heat stroke is characterized by extremely high core (rectal) temperature (106°–110° F), usually with profound coma, and usually in the *absence of sweating*. The onset of heat stroke is customarily dramatically sudden, with collapse and loss of consciousness. Convulsions may occur. Death may ensue rapidly.

Treatment consists of lowering of body temperature as rapidly as possible. As soon as the patient can be reached and a tentative diagnosis made, he should be rushed to the hospital or sickbay with the greatest dispatch and placed in a tub of ice water with ice chips or cubes in it. If cooling is started very early, the prognosis is far better than if it is delayed for an hour or more. The tub cooling may be halted when rectal temperature reaches 102° F, and the patient may then be placed in bed in the coolest area available. Rectal temperatures should be taken every ten minutes during tub cooling.

Obviously, such a severely ill patient must be kept under observation for days or weeks, and the Flight Surgeon should be in no hurry to return the aviator to duties involving flying, whether in actual control of aircraft or not. When the patient has been asymptomatic for a week or so, a test exposure to ambient temperatures, ***under close observation,*** should be considered to attempt to evaluate the normalcy of the patient's heat-regulating mechanisms. In the absence of evidence of a completely normal heat-regulation apparatus, the aviator should remain grounded.

Local manifestations of heat injury are limited primarily to the skin. Miliaria ("prickly heat") is an irritation of the sweat glands occurring in susceptible individuals who have been sweating profusely. Intertrigo is characterized by a moist, painful chafing of opposing skin surfaces. Hot, wet, macerated feet may resemble epidermophytosis. Most of these skin conditions may be controlled by good personal hygiene.

For the aviator, the most deleterious local manifestation of heat injury is sunburn, probably the most common of all head injuries. The dilated skin capillary beds resulting from sunburn actually decrease the acceleration tolerance of the aviator to a measurable degree. The interference with sleep resulting from the pain of a severe sunburn may decrease the alertness of the aviator and increase his accident potential. However, in the main, the aviator does not seek medical help from the Flight Surgeon despite severe sunburn, because he believes that some sort of lecture, or temporary grounding or other unpleasantness, will ensue as the result of his folly in being burned. To some extent, this is true, but recovery is rapid and grounding is not necessary for more than a few days.

Such conditions are usually detected by the Flight Surgeon who makes it a practice to frequent All-Pilots Meetings, prelaunch briefings, and other pilot meetings. The intelligent aviator grounds himself officially or unofficially in this situation, but the marginal pilot will attempt to fly to cover up for his bad judgment in getting sunburned.

As noted in the chapter on vision, night vision is adversely affected by the sunlight exposure which is usually necessary to obtain a severe sunburn.

Acclimatization

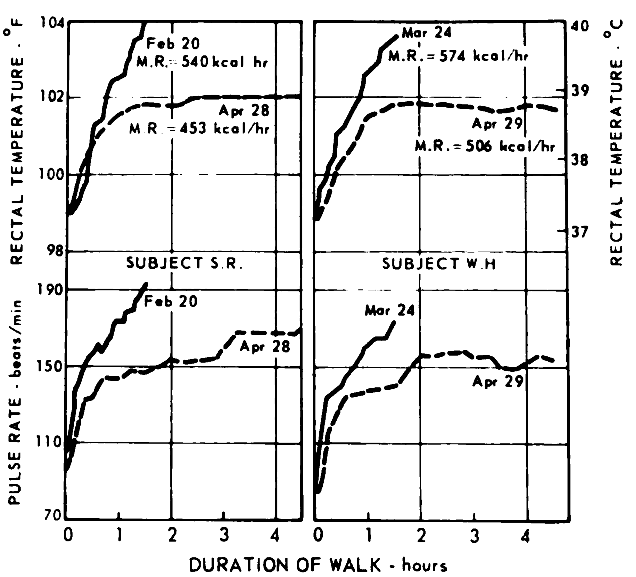
Acclimatization has been defined as a state of successful physiological adaptation of the individual to new climatic conditions. Although true physiologic adaptation to a hot environment was well established many years ago, the existence of cold acclimatization in man has been neither universally accepted nor demonstrated.

Adaptation to a hot environment begins immediately upon exposure to the environment. The adapting process continues with the exposure and is completely developed in four to seven days. Personnel can achieve an optimum level of adaptation, however, only if they engage in physical activity in the heat. Inactivity will result in very limited adaptation, even in extreme exposure. As mentioned previously, length and extent of activity should be increased gradually during the adaptation period, according to the table in NAVMED 5052–5. The effects of acclimatization are indicated in figure 12–6.

Heat Protection

Protection of personnel from the effects of heat will usually be successful through adherence to the following guidelines:

1. Maintain water balance. Failure to replace completely the water lost in sweat, hour by hour, leads to elevation of body temperature and excessive physiological strain. *Thirst is unreliable as an indication of the requirement for water intake to make up for heavy sweating.*



These data dramatically illustrate acclimatization to heat as shown by the lowering of body temperatures and heart rates of two men walking at 3.5 mph on a 5.6 percent grade in room temperature of 104° F, vapor pressure 13mm Hg (E. T. 84° F).

(1) Subject S. R. was acclimatized by 23 exposures to these conditions between February 20 and March 20. After March 20 his only exposures were on April 16 and 28.

(2) Subject W. H. was acclimatized by 11 exposures between March 24 and April 8. After April 8 his only exposures were on April 22 and 29.

On the first exposure, the experiment was terminated by the collapse of the subjects at 90 minutes. After acclimatization the men were still maintaining equilibrium with ease after 4.5 hours.

FIGURE 12–6. *Improvement from acclimatization* (BLOCKLEY, 1964; SOURCE: ROBINSON ET AL.).

2. Maintain salt balance. Mealtime replacement is adequate in contrast to the water situation above.
3. Regulate physical activity to a level consistent with avoidance of strain, which may be indicated by dizziness, breathlessness, and pounding heart.
4. Allow approximately 4 to 7 days for personnel to acclimatize. Increase the workload gradually over that period.
5. Provide work/rest cycles to allow the body to dissipate heat stored during periods of activity.
6. Wear light clothing and head covering which does not impair evaporation but does protect against heat absorption from the surroundings.
7. Ventilate and dehumidify indoor working areas. Air cooling may be employed also but is frequently less desirable than adequate air movement and humidity control.

THE PROBLEM OF WATER

Normal Operations

Water Intake.—For hot climates, both tropic and desert, with unrestricted water, figure 12–7 shows the distribution curve for the voluntary intake of a number of men performing medium work. The desert environment had air temperatures as high as 110° F and dry; the tropical air was mostly in the mid-80s, with a vapor pressure of 20–25mm Hg.

For planning purposes, the water requirements for personnel performing light, medium, and heavy work, in jungle and tropics, under both moderate and severe conditions, are detailed in the table contained in NAVMED 5052–5. In it, too, are schedules of work for personnel to achieve acclimatization to hot environments in the shortest possible time (six days), along with recommendations as to clothing, leadership requirements, and work scheduling in hot environments.

Any Flight Surgeon assigned to a unit located in, or scheduled for deployment to, a hot climate should study NAVMED 5052–5 carefully, because it contains a compendium of knowledge concerning the entire range of problems likely to be encountered in normal hot weather operations.

An important point to remember is that water intake must balance water output, and the total *amount* of water intake is otherwise immaterial.

Water Output.—Figure 12–8 gives sweating rates where the dry bulb temperature in the desert is constant and the amount of clothing in sun or shade,

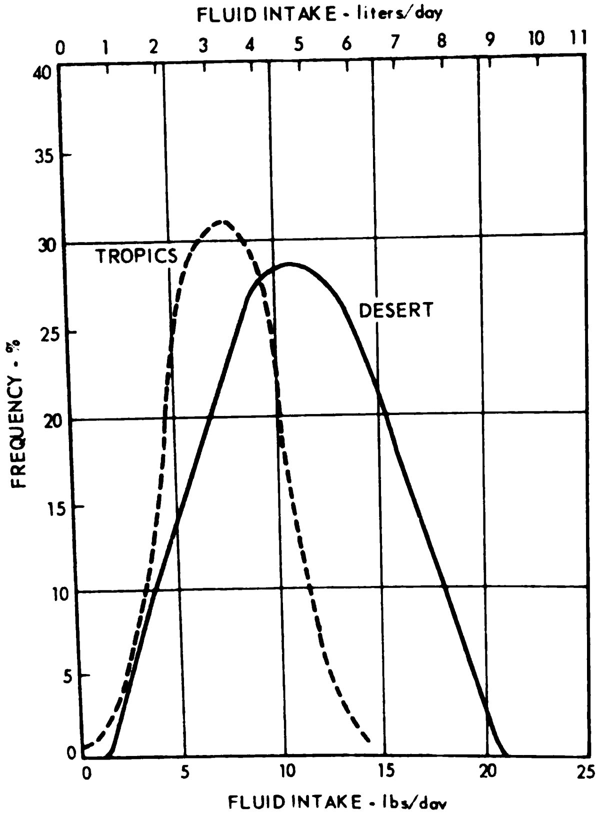


Figure 12–7. *Human daily fluid intake in tropics and desert* (KANTER & WEBB, 1964; ADAPTED FROM ADOLPH).

as well as the amount of work, are variables. Figure 12–9 gives sweating rates in lb/hr at various dry bulb air temperatures for variable amounts of work.

As man becomes dehydrated, the water loss via the kidneys also decreases. Figure 12–10 gives the quantities of urine output for various states of dehydration.

Most of the body's insensible water loss in hot climates is from extracellular water in the dermis which diffuses through the skin. Scott (1966) states that this route provides a rather constant heat loss rate at temperatures lower than 84° F, but that it rises rapidly above that point.

The amount of water which may be lost via skin diffusion is dependent on the vapor pressure gradient, skin temperature, and barometric pressure. Figure 12–11 shows the rates of water loss associated with different values

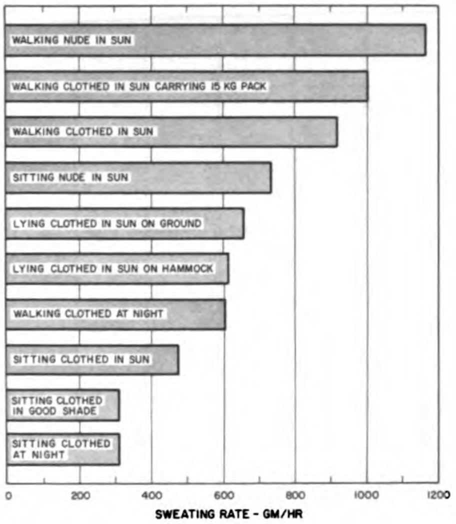


FIGURE 12–8. *Sweating rates are shown for various activities in the desert at an air temperature of 100° F dry bulb* (ROTH, 1964; SOURCE: ADOLPH).

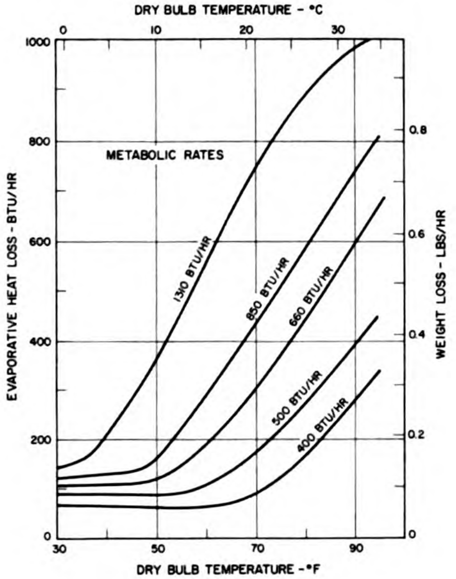


FIGURE 12–9. *The levels of sweating and evaporative heat loss expected at various temperatures are shown for activity levels ranging from sedentary to moderately hard work* (ROTH, 1964; SOURCE: THOMPSON RAMO WOOLDRIDGE).

Urine flow decreases progressively during dehydration, but probably never goes to zero even in severe dehydration. The data points shown by open circles are from one subject dehydrated seven different times by exposure to an air temperature of 122° F for eight to ten hours without food or water. The closed circles with bars show the means and standard errors of the mean for 13 subjects exposed to 122° F without food or water.

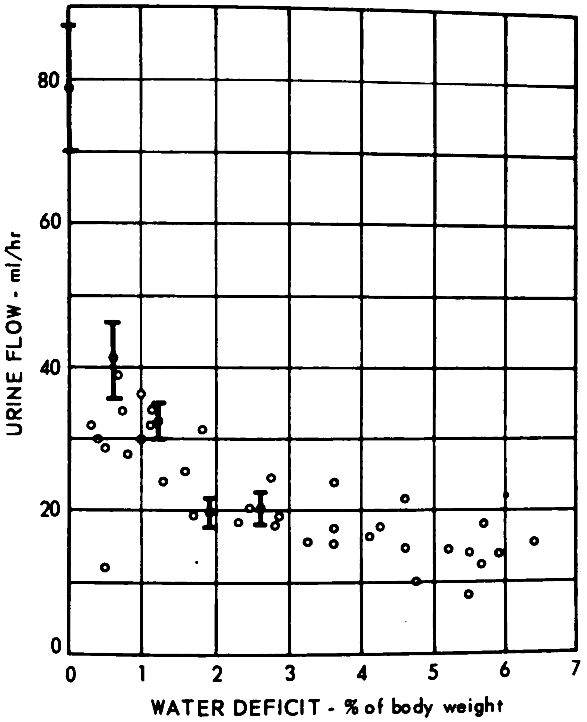


Figure 12–10. *Urine output for various states of dehydration.* (KANTER & WEBB, 1964; SOURCES: ADOLPH; & KANTER.)

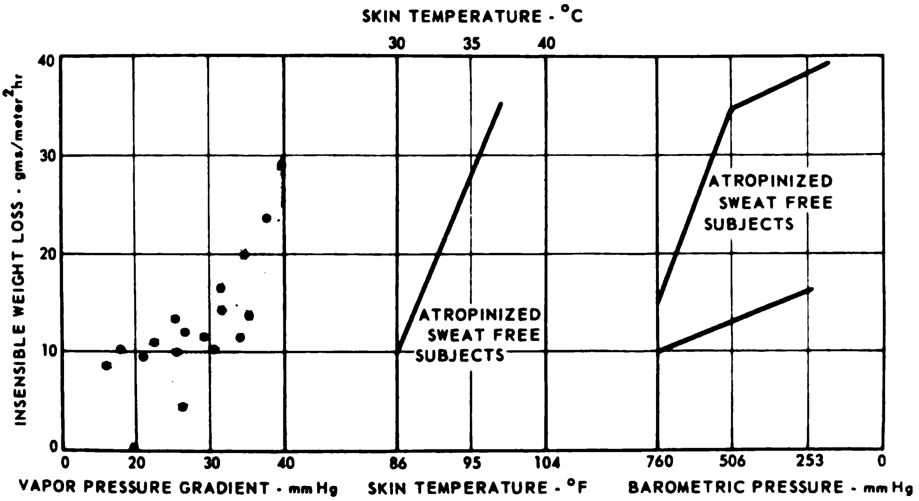


Figure 12–11. *Rates of water loss associated with different values of vapor pressure gradient, skin temperature, and barometric pressure.* (KANTER & WEBB, 1964; FROM DATA OF BREBNER ET AL.; HALE ET AL.; WEBB ET AL.; & ZOLLNER.)

of these variables. Figure 12–9 relates sweating and insensible loss to ambient air temperatures and work rates, with normal water intake.

Respiratory water loss is relatively constant for a fixed ventilation rate and for a fixed relative humidity. It may vary from 20 ml/hr resting (in dry air) to 200 ml/hr working heavily (in dry air).

Sea Survival

Historically, the story of maritime survival has been the story of procurement and effective utilization of water. In warm waters, man can survive for *months* with very little food, but only for *days* without water. Only a limited amount of canned emergency water is (or should be) carried in aircraft. Major effort over the past 25 years has been devoted to development of means for producing potable water from sea water, either by use of a solar still or water-desalting kit. While neither method is perfect, they can significantly increase survival time under many conditions.

Current emphasis in sea survival situations is directed toward improving means of detection and rescue of downed aviators with a view toward shortening the survival episode to the point where the water problem will no longer exist. Improved manual and automatically actuated radar reflectors are being developed and procured along with automatically actuated emergency radio beacons; and methods for open-sea rescue are continually being refined. This philosophy is paying enormous dividends in the types of survival situations presently being encountered.

Should requirements arise for fighting enemy forces far from land areas, however, open-sea survival problems—particularly the water problem—will again be of paramount importance.

Survival, in general, may depend more upon the mental attitude of the individual than upon the amount of water he has (or can get) or any of the other physical variables of the survival situation. The *Survival Training Guide* stresses this point. Every Flight Surgeon should be thoroughly familiar with the contents of this excellent publication.

Mental attitude toward survival has been discussed by a Navy psychiatrist at the Naval Aerospace Medical Institute. Tucker (1966) states that "Survival is something you do for yourself," and that some people simply give up and die rather than do it. Technically, he feels that the mental problem is one of stimulating aggression by leadership, either external or internal, and then directing that aggression outward to solve survival problems.

Causes of Dehydration.—In the earlier section on immersion hypothermia, Beckman's (1966) study of experimental subjects exposed to immersion for 12 hours at 75° F while wearing the MK-2 lifevest was mentioned. He found that a severe diuresis occurs; table 12–1 shows the values obtained. The body water loss which occurred during the mean immersion duration of 8.1 hours was about two percent, but the loss was directly related to duration of exposure.

Factors governing water output when the survivor is in a raft are essentially those outlined earlier. Reference to the tables provided will allow estimates to be made of water output under the particular survival conditions in the given environment.

Severe vomiting may result from seasickness, ingestion of petroleum products on the water's surface, or from accidental ingestion of sea water. In one experimental study performed in tropical ocean areas, seasickness alone caused vomiting for two or three days, resulting in termination of some subjects from the study even in the presence of unlimited amounts of fresh potable water (Dupree, 1958).

TABLE 12–1. *Mean Urinary Excretion Rates Before, During, and After Immersion to Neck Level in Water at 75° F*

Measurement	Number of subjects	Control	Immersion period			Postimmersion	
			2–4 hours	4–8 hours	8–12 hours	24 hours	48 hours
OUTPUT	5	89.2	211.4			26.9	36.8
ml/hr	10	59.0	179.3	84.0		39.9	42.8
	9	48.8	233.5	102.7	49.2	42.4	44.5
OSMOLARITY	5	49.8	107.4			23.6	31.1
Mosm/hr	10	42.5	129.6	48.8		34.6	38.1
	9	37.5	106.6	57.1	27.3	34.9	36.3
SODIUM	5	9.7	20.5			4.6	5.9
meq/hr	10	8.4	21.5	12.1		5.2	6.4
	9	8.6	21.3	12.8	7.1	7.1	4.5
POTASSIUM	5	6.6	13.0			1.7	2.3
meq/hr	10	2.9	8.2	3.8		2.6	2.7
	9	2.6	6.6	4.4	2.6	2.3	2.4
CALCIUM	5	0.36	0.58			0.27	0.32
meq/hr	10	0.38	0.21	0.08		0.13	0.30
	9	0.36	0.44	0.13	0.11	0.11	0.11

(Beckman and Reeves, 1966.)

If no solar still, desalting kits, emergency water supplies, or rainwater are available, dehydration will of course occur. Each desalting briquet will produce about one pint of potable water (which is reported to be barely palatable). The solar still will produce about one pint of palatable water per six hours of sunshine on a bright day.

Effects of Dehydration.—Figure 12–12 shows the effect of dehydration reported as percent initial weight, determined by several different sources. It should be clearly understood that the physiological effects of dehydration are combined with the psychological effects of the survival situation and with the physical stimuli of the environment, and the net result must be attributed to the combined stresses. Nevertheless, the figure may be of some value in teaching.

Expected Survival Times.—Figure 12–13 presents predicted survival times for both land and sea survival situations, with several conditions of water availability. However, these times can vary widely due to other variables, such as body surface area, skin thickness, and amount of protection afforded from the sun.

Means of Extending Available Water Supply.—A continuing problem in planning for and teaching survival procedures to aviation personnel is the presence of major misconceptions the student may have acquired in childhood and never questioned as to validity. When approaching the problem of water supply in the raft survival situation, it is good practice to question the students before making any comments in order to determine what prior misconceptions exist. One common misconception is that in the given situation, the principal objection to drinking urine is aesthetic. The *actual* reason is that it not only serves no useful purpose but accelerates the development of intracellular dehydration by introducing excessive electrolyte into the body for the amount of water involved. In fact, one might as well drink sea water.

Why not drink sea water? The best reason is that it will prove *fatal.* It introduces a hypertonic solution into the circulation, causes intracellular water to move into the extracellular space, and thus throws a load on the kidney to remove the excessive water. While the increased electrolyte is partially removed by renal filtration, the body experiences a net gain in electrolytes which causes a constant cellular space dehydration which must eventually cause death.

Investigations performed in the mid-1950s suggested that the train of events above might not be so serious as previously thought—that for three or four

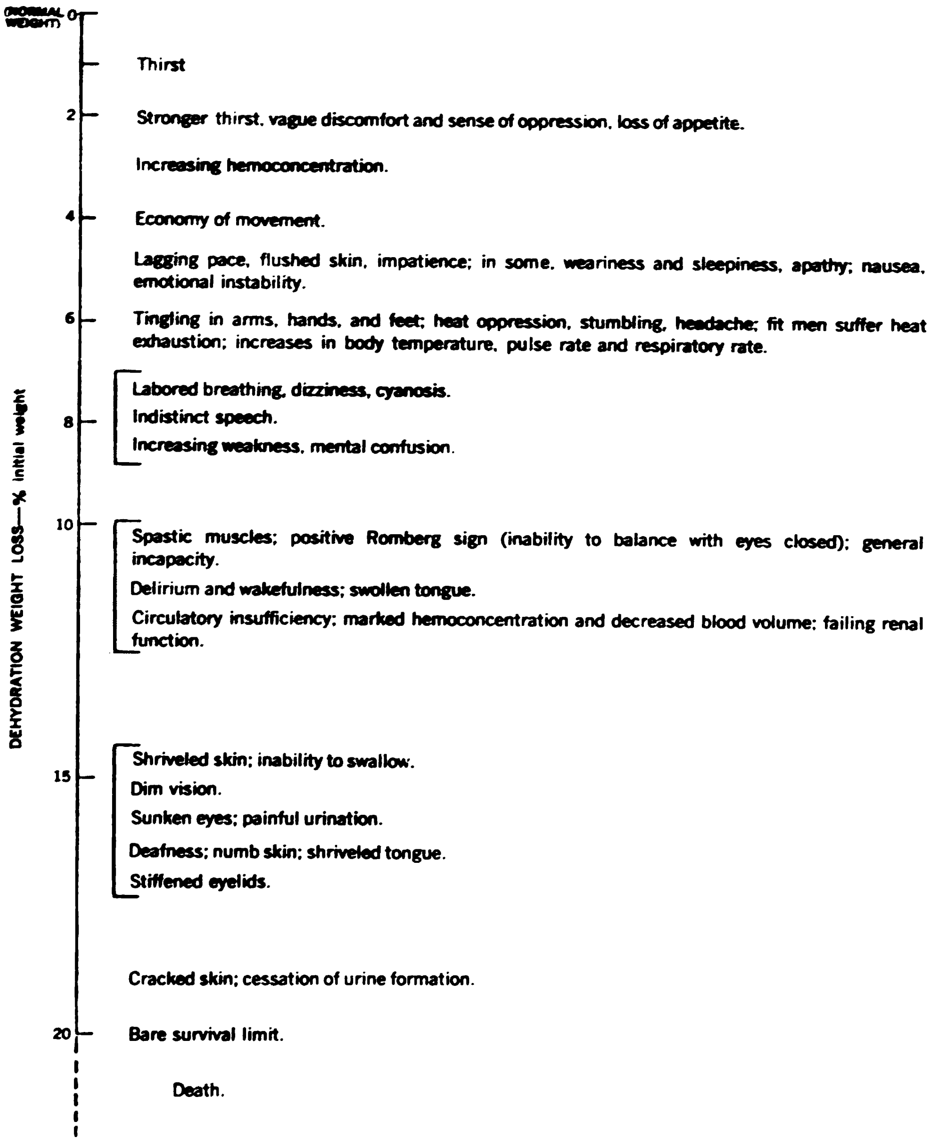

(NORMAL WEIGHT) 0
DEHYDRATION WEIGHT LOSS—% initial weight
Thirst
2
Stronger thirst, vague discomfort and sense of oppression, loss of appetite.
Increasing hemoconcentration.
4
Economy of movement.
Lagging pace, flushed skin, impatience; in some, weariness and sleepiness, apathy; nausea, emotional instability.
6
Tingling in arms, hands, and feet; heat oppression, stumbling, headache; fit men suffer heat exhaustion; increases in body temperature, pulse rate and respiratory rate.
Labored breathing, dizziness, cyanosis.
8
Indistinct speech.
Increasing weakness, mental confusion.
10
Spastic muscles; positive Romberg sign (inability to balance with eyes closed); general incapacity.
Delirium and wakefulness; swollen tongue.
Circulatory insufficiency; marked hemoconcentration and decreased blood volume; failing renal function.
Shriveled skin; inability to swallow.
15
Dim vision.
Sunken eyes; painful urination.
Deafness; numb skin; shriveled tongue.
Stiffened eyelids.
Cracked skin; cessation of urine formation.
20
Bare survival limit.
Death.

FIGURE 12–12. *Dehydration effects.* (KANTER & WEBB, 1964; SOURCES: ADOLPH; BEESON & MC DERMOTT (EDS.); GOLDBERGER; & CHANCE VOUGHT AIRCRAFT.)

days sea water might be consumed with impunity; and that for longer periods sea water diluted with fresh water might be utilized. This work caused a considerable international stir and resulted in the convening, by the World Health Organization, of a meeting of internationally recognized experts in

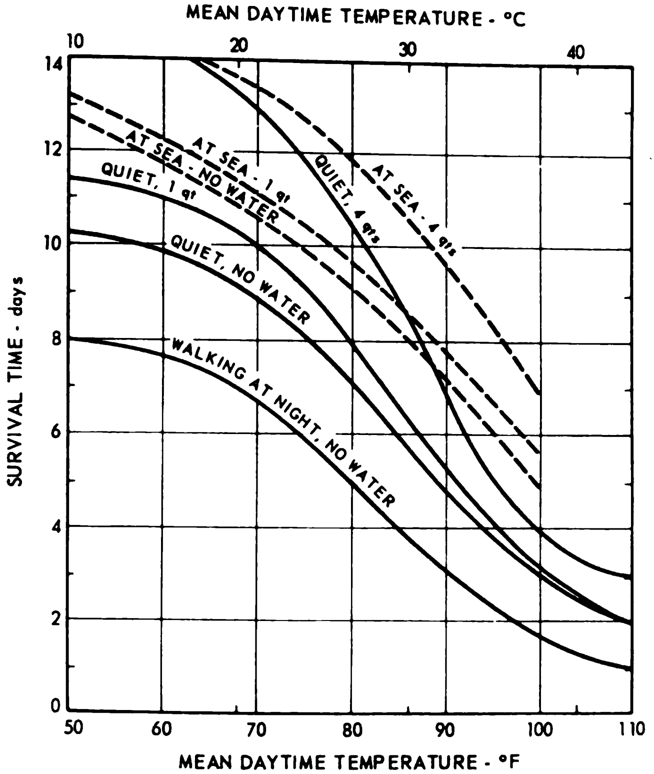


FIGURE 12–13. *Water intake and survival.* (KANTER & WEBB, 1964; ADAPTED FROM ADOLPH; NUTTALL; & U.S. AIR FORCE.)

Geneva to consider the question. Their report is published in the *WHO Chronicle* (1962).

These experts reached the following conclusions:

Effects on the Bowel: It was found that because of the cathartic effect of salts, drinking sea water is likely to lead to intestinal discomfort if not frank diarrhea. This effect is subject to individual variation and is more likely to occur if large amounts of sea water are ingested.

Effects on the Body as a Whole: The effect of drinking sea water is to overload the circulation with salt which can only be excreted by drawing upon the body water and so causing even greater dehydration.

Effects on the Mind: Drinking even small amounts of sea water affects some individuals unfavorably, while drinking large amounts leads to mental disturbances and even suicidal impulses.

Regarding dilution of sea water with available fresh water, the experts noted that it has been shown experimentally to be feasible in animals and is theoretically possible in man. But no acceptable evidence has ever been adduced that for man this is a satisfactory procedure. This as-yet-unproved hypothesis *cannot,* therefore, be offered as a practical recommendation for saving the lives of people in sea survival situations.

In essence, the committee stated: NEVER DRINK SEA WATER or mix sea water with fresh water if the latter is in short supply. NEVER DRINK URINE. DO NOT USE SEA WATER TO MOISTEN THE MOUTH, because the temptation to swallow it may be irresistible.

Suggestions have been made that if fresh-caught fish were pressed and the juice resulting were drunk, the fresh water supply could be supplemented. Hervey (1956) stated the now-accepted view that it seems unlikely that fish fluid can make a useful contribution to the castaway's water supply. The only way to obtain the fluid is to have a heavy press available which, in the survival situation, is most unlikely. Furthermore, fish fluid is high in protein content, and the net result is that all the water of the fish is needed in order to remove the urea.

The WHO Expert Committee recommended that survivors drink *no* water for the first 24 hours adrift. Then one pint of water daily per person should be taken until the supplies run low; then 100 ml/day until the supply is gone. *Note:* If great care is taken of the solar still and if the sun shines, this amount (one pint) or more per day can be produced almost indefinitely. Therefore, there is no reason to attempt any other means of supplementing the water available other than by drinking rainwater.

In the desperate situation which occasionally occurs in sea survival episodes, almost anything will be tried to alleviate thirst. Experiments in which sea water was instilled into the rectum and colon during water deprivation resulted in *greater* water *loss,* and increase in the osmotic pressure of the body fluids because of chloride absorption.

As noted previously, both sweat production and insensible water diffusion through the skin are directly related to skin temperature. In order to minimize these sources of evaporative water loss, the skin must be kept cool. This can be done by (1) erecting a barrier between the sun and the body, such as a parachute cloth parasol or awning, (2) avoiding unnecessary exercise and, thus, increased skin blood flow and sweat production, (3) directing whatever breezes exist onto the skin, (4) keeping cloths dampened with sea water on the skin to allow evaporative cooling from other than body water, and (5) occasionally completely immersing the body in the sea. As Whittingham (1965) points out, complete immersion is somewhat dangerous because a weakened man may not be able to reboard the liferaft.

Reduce vomiting loss by (1) taking seasickness preventive medications early, (2) avoiding ingestion of petroleum products during the initial phase of the survival situation, (3) not drinking fresh water at all until vomiting has completely ceased, and (4) spitting out and not swallowing, *if possible,* any sea water accidentally ingested during the initial phase of the survival situation.

Land Survival

General.—Water supply in jungle areas usually is limited only by its impurity. In arctic survival, again there is usually adequate water. Major problems, however, arise during desert survival. Earlier comments concerning the psychological aspects of survival at sea are equally applicable to survival in the desert. The Flight Surgeon can render a great service to aviation personnel by making the stark physiological facts of desert survival known to them.

Causes of Dehydration.—The water loss suffered during desert survival is far worse than that in sea survival because of the extremely low humidity, good conductivity of the soil, and the absence of convection currents in the desert. The heat load from radiation is considerably increased and that from conduction even more, because of the vast heated body of sand.

Sweating rates in the desert, under conditions of normal water intake, for various activities are given in figure 12–8. However, sweat rates for conditions of low as well as normal fluid intake are noted in figure 12–14. From the figure it can be seen that if fluid intake is extremely restricted, sweat rates will be low also, which means that the body's heat load from conduction and radiation may exceed the body's cooling capability, resulting in a net body heat gain with inevitable heat injury or death.

Figure 12–10 shows the urine outputs to be expected for given levels of dehydration, but these studies were conducted on human subjects and were terminated long before the subjects were completely incapacitated. The amount of urine output in such a near-terminal state of dehydration would be quite low.

Reference to figure 12–11 shows that in the extremely dry desert air, where the water vapor pressure gradient is rather high, skin diffusion water

In this chart, the fluid intake for men in the desert is seen to vary directly with sweat output. Ninety-one observations were made of 24-hour fluid intake and urine output, and the difference was taken to be sweat loss. Since the urine volumes were low, about 1.5 lbs. or 0.7 liters per man-day, the fluid intake matches rather closely the high sweat rates in the hot dry climate.

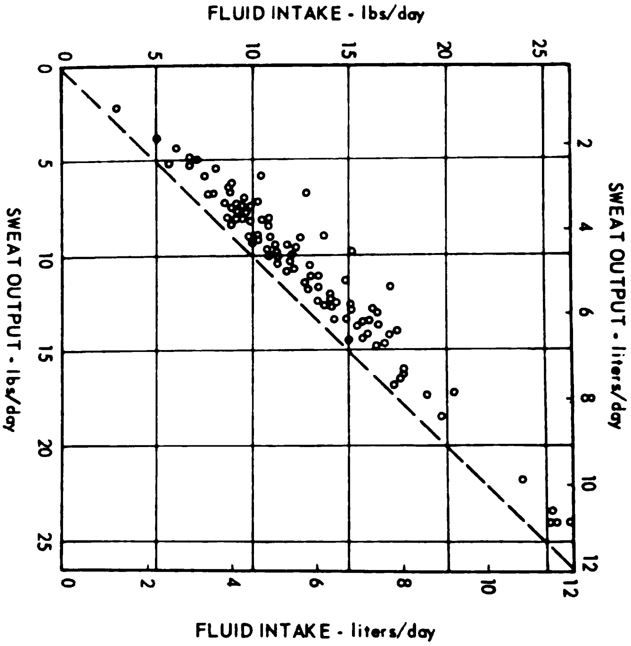


FIGURE 12–14. *Sweat rates for conditions of low, as well as normal, fluid intake* (KANTER & WEBB, 1964; ADAPTED FROM ADOLPH).

loss may be the major source of loss. An additional cause of skin diffusion loss is that skin temperature goes up, because of the increased heat gain from conduction and radiation, and because of decreased heat loss from diminished sweating.

Pulmonary insensible water loss is directly related to humidity and to ventilation rate. In the desert, the humidity is very low and thus there is a high pulmonary insensible water loss rate. If exercise (such as walking) is performed, the ventilation rate goes up and so does the water loss rate, perhaps by a factor of ten in the most severe situation where the air is dry and the highest ventilation rates occur as in performance of heavy work.

Desert Survival Expectancies.—Figure 12–13 presents much the same data arranged graphically. Note that these data are *averages* and exceptions certainly occur, as noted in the basic reference, the *Survival Training Guide.*

Conserving Available Water Supply.—There are two approaches to conservation of life which, in desert survival, means conservation of the water supply. One is to decrease the heat load, and the other is to extend the available water.

When sitting shaded from the sun by clothing alone, the sweating rate according to figure 12–8 is reduced from around 750 gm/hr to about 500 gm/hr. If sitting clothed in good shade, it is reduced to about 300 gm/hr. Gamble (1951) found that *total* evaporative water loss rate (including sweat and insensible water loss) was six to nearly ten times greater for a nude subject in the sun than for a nude subject in the shade, or for a nude subject in the sun exposed to a breeze. Both the latter conditions had about the same rate of evaporative water loss, 0.6 gm/kg/hr which is the irreducible minimum. Therefore, existing shade should be utilized or an artificial shelter from the sun should be made, leaving it open to the breezes.

Pinto (1962) states that if one can sit on an improvised seat one foot above the ground, the temperature may be 30 degrees cooler. Similar advantage may be gained by digging a hole to get beneath the hot layer of sand. However, the exertion involved in the latter case might cause increased evaporative water loss which would render the procedure useless.

As one would expect, work causes increased skin temperature, increased ventilation rate, increased metabolic heat production, and increased sweating rate. All these things result in increased water loss as well as increased total body heat load; therefore, exercise should be avoided except when absolutely necessary.

Available water must be conserved. The following three points should be well noted:

1. Water should be kept in closed containers if possible. The low desert humidity and high air temperature result in rapid evaporation from open containers, which are also subject to spillage.
2. Use *all* the water available. If a disabled vehicle is on hand, drain the water from the radiator. It may not look attractive, but it can make the difference between life and death.
3. Try to maintain fairly normal hydration until the water is all gone. Table 12–2 shows that the increase in survival time (with no walking) is about one day per quart of water at 80 degrees F. That is, nine days with no water, 13 days with four quarts of water. But if dehydration becomes so severe as to cause mental aberration, remaining water may never be used and may even be discarded. On the other hand, too high a water intake causes an increased sweat rate (which is a waste of water) and increased urine output (which again is a waste).

The work of Adolph and associates (1947) has established man's ability to withstand the desert environment in terms of the amount of water available and in terms of physical activity. The results of this work are summarized in table 12–2.

Obtaining Water in the Desert.—A desert water still can be improvised from a sheet of wettable plastic (such as Dupont Tedlar, .001 inch (1 mill) thick) about six feet square; a container such as a gallon bucket; and, if available, four to six feet of ¼″ plastic tubing (Jackson & van Bavel, 1965).

A bowl-shaped hole is dug in the soil, about 40 inches in diameter and 20 inches deep. Additional excavation in the center is needed to keep the top of the bucket a few inches above the bottom of the pit but below the bottom of the plastic. The tubing tip is fastened to the bottom of the container and brought out of the hole. Then the sides of the hole are lined with cut section of any plant material available (especially cactus) which might contain water. Polluted water or body waste can be used also if placed in a trough around the sides of the hole.

The plastic film is then placed over the hole and a little soil put around the edges to hold the plastic in place. Next, a fist-sized rock is placed in the

TABLE 12–2.—*Days of Expected Survival in the Desert Under Two Conditions*

Condition	Max Daily Shade Temp °F	Total Available Water Per Man, U.S. Quarts					
		0	1	2	4	10	20
No walking at all, resting in shade.	120	2	2	2	2.5	3	4.5
	110	3	3	3.5	4	5	7
	100	5	5.5	6	7	9.5	13.5
	90	7	8	9	10.5	15	23
	80	9	10	11	13	19	29
	70	10	11	12	14	20.5	32
	60	10	11	12	14	21	32
	50	10	11	12	14.5	21	32
Walking at night until exhausted and resting thereafter..........	120	1	2	2	2.5	3	....
	110	2	2	2.5	3	3.5	....
	100	3	3.5	3.5	4.5	5.5	....
	90	5	5.5	5.5	6.5	8	....
	80	7	7.5	8	9.5	11.5	....
	70	7.5	8	9	10.5	13.5	....
	60	8	8.5	9	11	14	....
	50	8	8.5	9	11	14	....

(Adapted from *Physiology of Man in the Desert* by E. F. Adolph and Associates, copyright 1947. Used by permission of John Wiley & Sons, Inc.)

center of the plastic sheet and pushed down until it is about 15″ below the surface of the ground, forming an inverted cone. The cone must have an angle of about 30 degrees in order for the water drops to run down into the container. Soil is placed on the plastic all around the rim to hold it securely in place and prevent water vapor losses. The tubing is brought out from under the plastic sheet to be used for drinking the water from the container without disturbing the plastic sheet.

If cactus, wet sand, body wastes, or other source of water is used, yield is about three pints daily, but if only dry desert soil is available, no more than one-half pint per day can be expected. Sea water can be distilled in this way.

CAUTION. Do not let contaminated soil or plant material touch the plastic because it will cause contamination of the water. See figure 12–15 for diagram.

Detection and Rescue

When in a survival situation, or when such a situation is imminent, the victim must be prepared to use all techniques and equipment at his disposal to facilitate the search and rescue (SAR) process. This preparation involves a knowledge of the SAR organization; the types of SAR vehicles; signaling equipment; and recovery techniques and equipment. These areas are extensively covered in the Navy's *Survival Training Guide,* NAVWEPS 00–80T–56, and *Safety and Survival Equipment Manual,* NAVAER–00–80T–52.

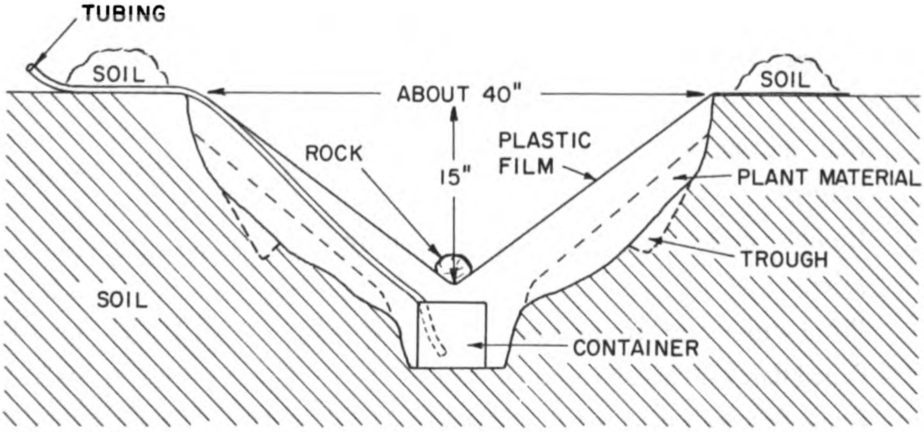


FIGURE 12–15. *Improvised desert water still* (Crossfeed, SEP. 1965).

The following discussion is intended to provide the Flight Surgeon with some background by highlighting the detection and recovery portions of the SAR process.

Preparation.—Preparation for survival begins before the flight when mission participants receive their briefing, which normally includes a search and rescue review. Coverage is typically brief but comprehensive, with emphasis on location and capabilities of SAR units in the operating area; review of downed wingman procedures; listing of emergency communication channels; positions of friendly and hostile forces; and a review of emergency equipment. Preparation for a potential survival situation is continued by the crew when they conduct a preflight inspection of their survival equipment to include presence and operability checks of signaling devices. Finally, when aircraft abandonment or ditching is imminent, the pilot (or crew) must convey knowledge of his predicament to a wingman, ground forces, or by communicating in the blind, so as to activate the SAR process in a timely fashion.

Signaling Equipment.—The pilot and his aircraft are equipped with a full array of normal and emergency communication equipment. Survival officers and flight equipment officers of operating units are responsible for training aviation personnel in the capabilities, limitations, and operating characteristics of these equipments as well as those which might be airdropped to a survivor. In addition, the aviator should be familiar with SAR procedures so that he can assess his situation and not expend his signaling devices fruitlessly. The Navy manuals referenced above provide complete descriptions of all emergency signaling equipment currently in use or available to operating squadrons. A listing of the types of devices is provided here:

Flare and smoke devices for night and day.

Whistle.

Lights, battery-powered, steady and flashing.

Mirror.

Pistol with tracer ammunition.

Pencil flare gun MK 79 Mod O.

Radio beacon transceivers.

High-visibility colors and signaling panels.

Dye markers for snow and water.

Radar reflectors.

SOFAR bombs (sonar signaling depth charges).

Sonobuoys can be used as transmitters from water to ASW aircraft.

Signaling techniques using arm motions, ground-marking symbols, or fires are of course available and are described in the referenced manuals.

Recovery.—Survivors and recovery crews must be knowledgeable and proficient concerning the capabilities and limitations of recovery techniques and equipment so as to minimize potential hazards. There are many variables in the recovery process but the primary factors are the type of recovery vehicle, its recovery equipment, the physical condition of the survivor, and the environmental conditions. The Navy manuals referenced above provide a complete and current description of all techniques and equipment.

The helicopter and plane guard destroyer are the two types of vehicles most frequently employed for the recovery portion of the SAR. A helicopter rescue can be made of a survivor in the water or in a liferaft, and, by utilizing rescue crewmen, of a totally incapacitated survivor. On land, the helicopter may land for recovery or, if the condition of the survivor permits, may accomplish a mobile (running) pickup, in which a running survivor grasps and ascends a line or ladder trailing from the slowly moving craft. Sea recoveries by helicopter normally employ a sling or seat-type recovery device. The former is a horsecollar affair which is lowered from the helicopter. The survivor places himself (or is placed) into the sling so that it fits under his arms, and faces the attaching bolt. When hoisted, the sling pressure is applied across the survivor's back, and the sling is locked under the armpits. The seat, when lowered from the helicopter, is an erect cylinder with three horizontal flanges. The survivor mounts the flanges and grasps the cylinder with his arms during the hoisting process.

Newer and simpler devices are being developed constantly to improve the U.S. Navy's survivor recovery capability. For example, an experimental skyhook system is being studied and tested. This system allows conventional aircraft, flying at moderate speeds, to snatch a prepared survivor from water or land. The Flight Surgeon is urged to keep abreast of these developments through interaction with survival personnel of his organization and through a regular review of Aviation Clothing and Survival Equipment Bulletins, which are issued on an as-needed basis by the Bureau of Naval Weapons.

MOTION PICTURE FILMS FOR SURVIVAL AND RESCUE

Number	*Title*	*Date*
	Rescue	
MG-4327	Air-Sea Rescue (18 minutes)	1945
MN-5309A	Search and Rescue—Visual Aspects of Search and Signaling (19 minutes)	1947
MN-6483A	Expendable Radio Sono-Buoy in Air Sea Rescue—Description	1946
MN-6483B	Expendable Radio Sono-Buoy in Air Sea Rescue—Procedures and Operations	1946
MN-8760A	Helicopter Rescue at Sea	1959
MN-8760C	Destroyer Rescue	1962
9565	Helicopter Rescue Operation	....
	Cold Weather Operation	
MA-7450	General Effects of Cold on Man	1951
MH-7491	Cold Weather Training—Indoctrination and Survival	1953
FA-7923	How to Use Cold Weather Clothing	1952
MA-8277	Frostbite Parts I and II	1954
MA-8851	Prevention of Cold Injuries	1958
	Survival	
MN-1329	Forced Down at Sea	1943
MN-7458B	Aviation Exposure Suit Mk-4	1953
MV-7818	Survival in the Aleutians	1951
MN-7918	Making Sea Water Drinkable with the Solar Still and Desalting Kit	1953
MV-8381	Survival on Polar Sea Ice	1954
MV-8384	Survival on the Ice Cap	1955
MV-8677	Sun, Sand and Survival	1954
MV-8826	Jungle Survival	1958
FN-9202A	Survival in the North Temperate Regions—Living Off the Land	1955
MV-9404	Survival Training Series:	
	C. Preparing an Animal for Use	1957
	D. Utilization of Game	1957
	G. Preparing Plant Food for Use	1957
	M. Medical Aid	1957
	W. Shelter	1958
9459	Mk-5 Anti-Exposure Suit	....
9781	Edible Plants of Field and Forest	....
9794A	Survival Stresses	....
9794B	Tropic Sea Coast Survival	....

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CHAPTER 13

Acceleration/Vibration Stress

ACCELERATION FORCES

- G-Force Syndrome
  - Linear Acceleration
  - Radial Acceleration
  - Angular Acceleration
  - Combined Acceleration
- Tolerance to Acceleration Forces
- Influence of Acceleration Forces on the Cardiovascular System
- Influence of Acceleration Forces on Sensory Modalities
  - Vision
  - The Proprioceptive Sense
  - Audition
- Influence of Acceleration Forces on Behavioral Characteristics
  - Motor Performance
  - Complex Tasks
  - Cognitive Processes
- Protection Against Acceleration Forces
  - Straining Maneuvers
  - Antiblackout System

VIBRATION

- Physics of Vibration
- Parameters of Vibration
- Vibrations in Aircraft
- Effects of Vibration on Humans
  - Impedance Studies
  - Subjective Tolerance Levels
- Effects of Vibration on Performance
  - Information Channels
  - Tracking

PATHOLOGY
THERAPY
  Protection from Vibration
REFERENCES

ACCELERATION FORCES

FLIGHT IN fixed-wing naval aircraft routinely subjects an aviator to accelerations and decelerations. When exposed to such forces, the body undergoes many changes, ranging from a complete collapse of the cardiovascular system to a simple increase in the fatiguing characteristics of flight. Related to the physiological changes produced by these stresses are corresponding changes in the performance capabilities of the pilot. For example, in certain protective clothing, under a high level of acceleration, the pilot may have great difficulty in activating the escape mechanism of his aircraft. Too abrupt a pullout from a dive can cause a pilot to lose his vision or even consciousness. Visual illusions of considerable magnitude can also be produced.

It is apparent that man is ill-equipped to withstand severe acceleration stresses. It also is apparent that in military flying such stresses will increase as the performance characteristics of aircraft increase. Therefore, flight personnel must understand those forces and factors which can either increase or decrease acceleration tolerance. Without such an understanding, on the part of both the Flight Surgeon and the pilot, many avoidable accidents will continue to take place and entries in the "cause underdetermined" accident category will continue.

G–FORCE SYNDROME

When a force is applied to a body so as to change its velocity or direction of movement, the effect due to inertia is one of force occurring in the opposite direction. Thus, when a vehicle is accelerated forward, the operator is "pressed back" into his seat because of the tendency of his body to remain in its preacceleration state. Similarly, when the vehicle is decelerated, the operator is forced away from his seat and into his restraining harness.

In specifying the direction of acceleration forces, two sets of terminology are commonly used—one refers to the direction of acceleration of a mass; the other refers to the inertial reaction of the tissues and fluids of the intact mammalian body in response to the acceleration applied to the whole body (see column 7 [system 2] and column 8 [system 3] of table 13–1). The measurement of the inertial resultant of whole body acceleration is stated in terms of G in multiples of the magnitude of the acceleration of gravity (G_0), or 32 feet per sec^2 (981 cm/sec^2). Classically, in aerospace medicine, three basic types of acceleration forces have been described as occurring in flight.

Linear Acceleration

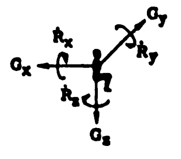
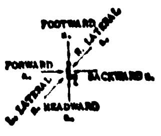
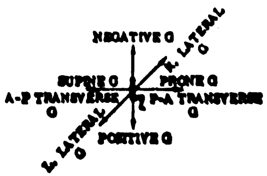
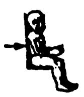
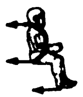
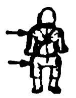
Linear acceleration is a progressive increase or decrease in velocity along a straight line. Linear acceleration in aircraft occurs primarily on takeoff and landing. In naval aviation, linear acceleration stresses are encountered in catapult launchings, carrier deck landings, during emergency ejections, crash landings, and ditchings. A high-speed carrier arrestment may exceed 6 G.

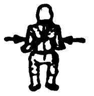
Radial (Centrifugal) Acceleration

Radial acceleration involves a change in direction but not necessarily any change in velocity of travel. Radial acceleration forces are produced by centrifugal force, tending to cause an object to continue in the same direction of motion upon the application of other forces causing it to change direction.

The inertial resultant of this type of acceleration on the body is the same as that shown in system 3 of table 13–1, depending upon the relationship of the body axis to the axis of rotation. Since the magnitude of radial acceleration forces is proportional to the square of the velocity and inversely proportional to the radius of the turn, it follows that as the speeds of aircraft incease, radial acceleration forces must also increase. It is not always feasible to increase the radii of turns to such an extent as would be necessary to maintain low-level radial G forces. However, the aerodynamic characteristics of modern aircraft impose certain limitations. At 40,000 feet, a jet cannot pull high-G turns without encountering buffet, a form of stall. In supersonic flight, higher G turns can be made, but deceleration occurs which results in the plane becoming subsonic and again encountering strong buffet.

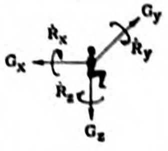
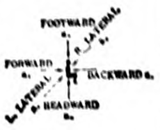
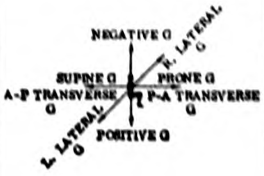
TABLE 13-1. *Physiological Acceleration Systems*[a]

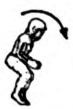
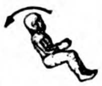
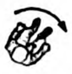
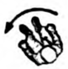
				System 1		System 2	System 3[b, c]	System 4	System 5
				 G_x $\dot{R}_x$ $\dot{R}_z$ G_z G_y $\dot{R}_y$		 FOOTWARD a. R. LATERAL a. FORWARD a. BACKWARD a. L. LATERAL a. HEADWARD a.	 NEGATIVE G R. LATERAL G SUPINE G PRONE G A-P TRANSVERSE G P-A TRANSVERSE G L. LATERAL G POSITIVE G		
Verbal definition [1]	Pictorial description [2]	Descriptive terms [3]	Vernacular description [4]	AGARD symbols[b] [5]	Heart displacement [6]	[7]	[8]	[9]	[10]
Linear forces or accelerations									
A force applied to the posterior part of the trunk, *acting forward with respect to the subject* and perpendicular to the mean spine *produces a forward acceleration*		Forward acceleration or forward acting force	Eyeballs in	$+G_x$	Moves toward back	Forward acceleration	Transverse A-P G Supine G Chest to back G	Sternumward	Surge
A force applied to the anterior part of the trunk, *acting backward with respect to the subject* and perpendicular to the mean spine *produces a backward acceleration*		Backward acceleration or backward acting force	Eyeballs out	$-G_x$	Moves toward front	Backward acceleration	Transverse P-A G Prone G Back to chest G	Spineward	-
A force applied to the left surface of the subject's body, *acting in a rightward direction* and essentially perpendicular to the subject's mean spine *produces a rightward acceleration*		Rightward acceleration or rightward acting force	Eyeballs left	$+G_y$	Moves toward left	Right lateral acceleration	Left lateral G	—	Left sway

A force applied to the right surface of the subject's body, *acting in a leftward direction* and essentially perpendicular to the subject's mean spine *produces a leftward acceleration*		Leftward acceleration or leftward acting force	Eyeballs right	$-G_y$	Moves toward right	Left lateral acceleration	Right lateral G	—	Right sway
A force applied to the buttocks, thighs, and/or feet, *acting in a headward direction with respect to the subject* and essentially parallel to the subject's mean spine *produces a headward acceleration*		Headward acceleration or headward acting force	Eyeballs down	$+G_z$	Moves toward feet	Headward acceleration	Positive G	Headward	—
A force applied to the shoulders, thighs, and feet of a seated human *acting in a tailward direction with respect to the subject* and essentially parallel to the subject's mean spine produces a *tailward acceleration*		Tailward acceleration or tailward acting force	Eyeballs up	$-G_z$	Moves toward head	Footward acceleration	Negative G	Tailward	Heave
				Oscillatory forces or acceleration					
Forces that alternate in direction and produce alternately forward and backward motion of the subject, and that act essentially perpendicular to the spine, produce *front to back acceleration*		Front-to-back oscillating force or acceleration	—	$\pm G_x$	Oscillates fore-and-aft within thorax	—	—	—	—
Forces that alternate in direction and produce alternately side to side motion of the subject, and that act essentially perpendicular to the spine, produce *side to side acceleration*		Side-to-side oscillating force or acceleration	—	$\pm G_y$	Oscillates side-to-side within thorax	—	—	—	—

(See footnotes at end of table.)

TABLE 13–1. *Physiological Acceleration Systems*[a]—Continued

				System 1		System 2	System 3[b, e]	System 4	System 5
				 G_x $\dot{R}_x$ $\dot{R}_z$ G_z G_y $\dot{R}_y$		 FOOTWARD a. R. LATERAL b. FORWARD a. BACKWARD a. L. LATERAL b. HEADWARD a.	 NEGATIVE G R. LATERAL G SUPINE G A-P TRANSVERSE G PRONE G P-A TRANSVERSE G L. LATERAL G POSITIVE G		
Verbal definition	Pictorial description	Descriptive terms	Vernacular description	AGARD symbols[h]	Heart displacement				
[1]	[2]	[3]	[4]	[5]	[6]	[7]	[8]	[9]	[10]
Oscillatory forces or acceleration—continued									
Forces that alternate in direction and produce alternately head to tail motion of the subject, and that act essentially parallel to the spine, produce *head to tail acceleration*		**Head-to-tail oscillating force or acceleration**	—	$\pm G_z$	**Oscillates head-to-tail within thorax**	—	—	—	—
Angular moments or acceleration									
***A rotational moment or couple* that produces a *head left motion* of the subject that lies essentially in the frontal (shoulder-to-shoulder) plane produces a *head left cartwheeling* angular acceleration**		**Head left cartwheeling moment or acceleration**	—	$-R_x$	**Top tilts toward right shoulder**	—	—	—	—
***A rotational moment or couple* that produces a *head right motion* of the subject that lies essentially in the frontal (shoulder-to-shoulder) plane produces a *head right cartwheeling* angular acceleration**		**Head right cartwheeling moment or acceleration**	—	$+R_x$	**Top tilts toward left shoulder**	—	—	—	—

A rotational moment or couple that produces a *head-forward feet backward* tumbling motion of the subject that lies essentially in the saggital plane produces a *forward somersaulting* angular acceleration		Forward somersaulting moment or acceleration	—	$-R_y$	Top tilts toward spine	—	—	—	—
A rotational moment or couple that produces a *head-backward feet-forward* tumbling motion of the subject that lies essentially in the saggital plane produces a *backward somersaulting* angular acceleration		Backward somersaulting moment or acceleration	—	$+R_y$	Top tilts toward sternum	—	—	—	—
A rotational moment or couple that produces a *right-turn motion* of the subject about the spine in the saggital plane produces a *right twisting* angular acceleration		Right twisting moment or acceleration	—	$+R_z$	Twists toward subject's left	—	—	—	—
A rotational moment or couple that produces a *left-turn motion* of the subject about the spine in the saggital plane produces a *left twisting* angular acceleration		Left twisting moment or acceleration	—	$-R_z$	Twists toward subject's right	—	—	—	—

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[a] The forces and accelerations to which vehicle occupants may be exposed are defined in column 1 and pictured in column 2. The descriptive terms to be used in discussions are tabulated in column 3 and the "eyeballs" vernacular terms are listed in column 4. The symbols previously recommended by AGARD and now to be associated with the terms in column 3 are listed in column 5. The reaction and motion of the heart *with respect to the subject's body* are listed in column 6. Previously used terminologies are listed in columns 7–10.

[b] Capital G is used as a unit to express inertial resultant to whole body acceleration in multiples of the magnitude of the acceleration of gravity, $g_0 = 980.665$ cm/sec^2 = 32.1739 ft/sec^2.

[c] A–P: anterior-posterior P–A: posterior-anterior.

(Allen, 1965; compiled by Gerard J. Pesman, Manned Spacecraft Center, National Aeronautics and Space Administration.)

Angular Acceleration

Angular acceleration occurs when the axis of rotation is through the pilot's body or very close to it (see column 2, table 13–1). Such accelerations occur during the banking of an aircraft going into a turn—also in spins. The tumbling during parachuting and upon emergency ejection also produces angular accelerations. This type of acceleration, even at small magnitudes, will disturb the functioning of the semicircular canals, producing vertigo and disorientation.

Combined Acceleration

In aircraft flight, usually more than one type of acceleration is encountered. For example, when an aircraft makes a turn, causing a change in direction, with a resultant radial acceleration force, the plane also is generally banked, which results in an angular acceleration force to the pilot. (Combined accelerations are also experienced during ejection, when a linear acceleration is experienced as well as a forward tumbling, with its resultant angular force.)

Oscillatory Acceleration

Oscillatory acceleration involves a rapidly alternating direction of motions such as occurs in buffeting and turbulence. These alternating forces and their symbols are shown in table 13–1. The table also gives presently accepted standard terminology, definitions, and symbols as used in the acceleration literature. In order better to understand his reading on acceleration, the Flight Surgeon should study this table in detail. In addition, he should be able to utilize the proper symbols and terms in his reports describing acceleration forces acting during various aircraft incidents and accidents.

TOLERANCE TO ACCELERATION FORCES

The effects of acceleration forces are typically described with regard to changes produced in perceptual-motor performance and physiological functioning. Physiological and performance tolerance limits often are functionally related. However, they are not necessarily identical since each depends upon the criteria applied. Furthermore, the determination of reliable criteria presents a problem in itself. Coburn, Beckman, and Duane (1963) discuss the difficulty in selecting an objective, easily discernible endpoint which would consistently indicate the circulatory state of the human central nervous system during exposure to positive acceleration. They present evidence which indicates "loss of ocular motility" to be a more reliable endpoint than either "grayout" or "blackout" which respectively represent subjective reports of partial or complete loss of vision. This measure as yet has not seen extensive use.

Rate of G development and time at maximum G, for positive acceleration conditions, to produce visual symptoms and unconsciousness are shown in figure 13–1. The curves of figure 13–1 show the decrease in G tolerance which occurs with an increase in the rate of onset of the acceleration force.

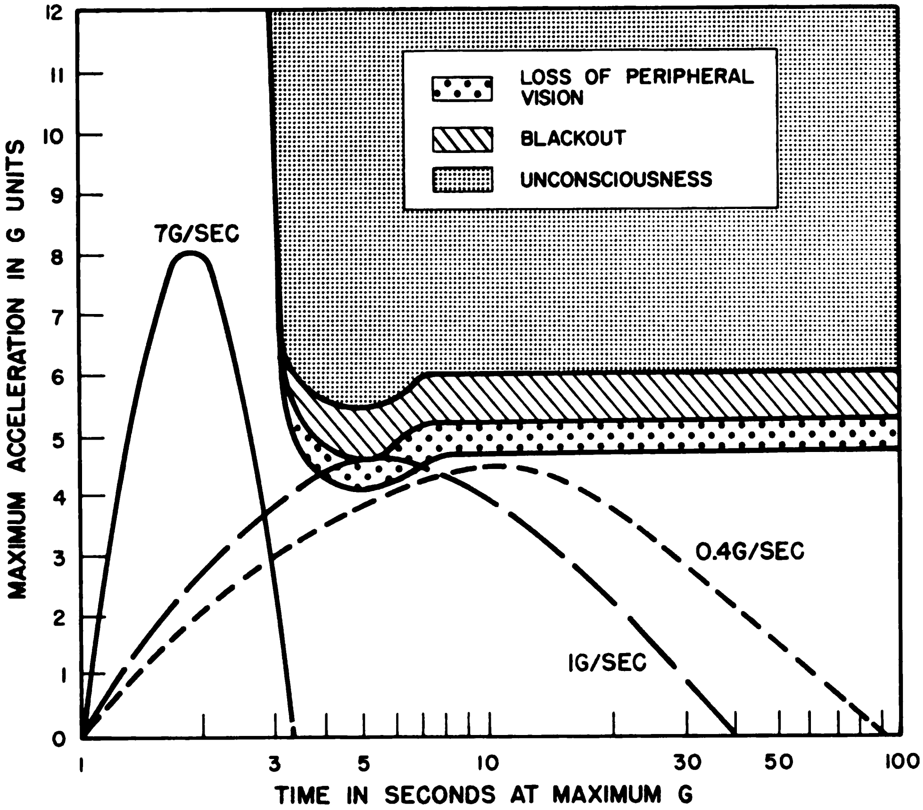

FIGURE 13–1. *Acceleration and time at maximum G required to produce visual symptoms and unconsciousness. Curves showing different rates of G development are given to demonstrate the importance of this parameter for the occurrence of loss of peripheral vision and blackout* (WHITE, 1961, USED BY PERMISSION OF LITTLE, BROWN AND COMPANY).

Figure 13-2 shows tolerance to negative acceleration, based on the limited data available. A direct comparison of tolerance curves for positive and negative acceleration cannot be made, however, since the negative tolerance curve indicates the combination of acceleration and time which an individual is prepared to endure rather than reflecting a more objective index, such as onset of unconsciousness.

INFLUENCE OF ACCELERATION FORCES ON THE CARDIOVASCULAR SYSTEM

The most obvious effect of a positive acceleration force is the increased weight of all parts of the body. Since weight is defined as the product of mass times acceleration, weight increases in direct proportion to acceleration. For example, at 4 G a 200-pound man effectively weighs 800 pounds. This same relationship exists internally. The weight of the normal blood volume is also increased by a factor of four.

Excessive gravitational stressing of the cardiovascular system causes gross disturbances in the local distribution of blood volumes and in the pressures

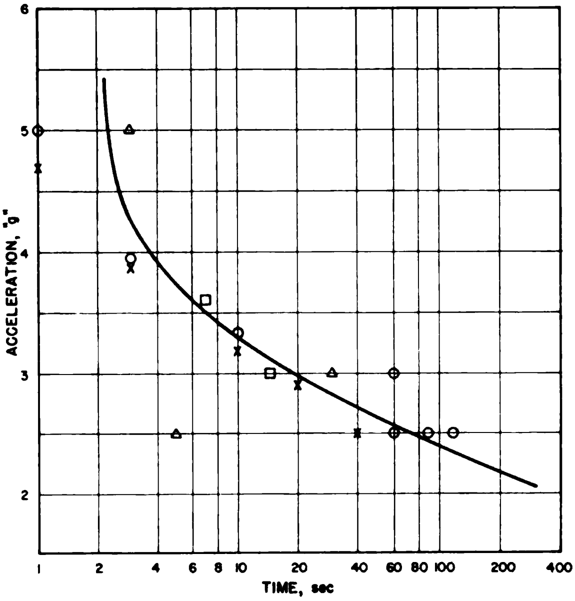

FIGURE 13-2. *Tolerance to negative acceleration from a number of sources* (HOWARD, 1965, BY PERMISSION OF THE CONTROLLER OF HER BRITANNIC MAJESTY'S STATIONERY OFFICE).

at which the blood operates. Most of the concomitant physiological and performance degradations undergone during periods of gravitational stress are a function of basic changes within the cardiovascular system.

Acceleration stresses produce the greatest pressures in those parts of the system that are farthest from the heart. Man is so constructed that when he is seated his heart lies approximately at the point of junction of the upper and middle thirds of a long cylindrical body. The head, which is the structure most sensitive to deficiencies in pressure, is located at one end of this cylinder and is approximately 30 centimeters from the heart. Normal peak arterial pressure is equal to 120mm Hg. At 5 G, a standing body column of 30 centimeters exerts a pressure of 120mm Hg, thus exactly balancing arterial pressure. Under such circumstances, either compensatory reflex mechanisms must come into play or blood flow through cranial arteries will cease. Reflexogenic areas which respond to changes in hydrostatic pressure may either help to counteract the force of acceleration or may, in fact, contribute to its deleterious effects. For example, carotid sinus reflex clearly enhances resistance to prolonged $+G_z$. However, when the same force is applied in the opposite direction ($-G_z$), as would be the case during an outside loop or spin in an aircraft, this reflex may seriously jeopardize a pilot's life by nearly stopping the heart (Gauer & Salzman, 1961).

Effects of transversely applied positive acceleration include a decrease in arterial blood oxygen saturation due to disturbance of pulmonary circulation (Hershgold, 1960; Wood, Nolan, Marshall, Cronin, & Sutterer, 1963); elevation of venous blood pressure in the thorax at the level of the superior vena cava and in the right atrium, and cardiac rhythm irregularities in the form of premature contractions often originating in the ventricle (Smedal, Holden & Smith, 1963; Nolan, Marshall, Cronin, Sutterer, & Wood, 1963). An additional effect of $+G_z$ acceleration is that of fluid accumulation and the pooling of blood in the abdominal veins and those of the lower extremities.

During $-G_z$ acceleration, high cranial pressures will develop which may be sufficient to rupture thin-walled capillaries (Gauer & Henry, 1964). This, in addition to the fact that body reflex mechanisms apparently are not as efficient for negative acceleration, limits tolerance to this type of acceleration vector. During $-G_z$ acceleration, vision may "redout," presumably because of enlargement of capillaries in the retina. This, however, is not completely confirmed experimentally, and it is thought instead that vision may be obscured by the forcing of the lower eyelid over the cornea by G forces (Henry, 1950). The muscles for this lid are weak and not designed to hold it in place against the force of negative gravity. Retinal hemorrhages have never been observed in man during experimental exposures to $-G_z$ acceleration on centrifuge.

INFLUENCE OF ACCELERATION FORCES ON SENSORY MODALITIES

Vision

The general effects of acceleration on vision are described by investigators at the Aerospace Medical Research Department as follows:

"During exposure to high positive, negative, and transverse acceleration, visual disturbances occur. During positive acceleration, these disturbances result primarily from ischemia; however, mechanical distortion of the eye may also occur in severe cases. Generally, a period of grayout exists before blackout occurs. Grayout is characterized by general dimming and blurring. Total visual disturbance occurs approximately one G unit below the level at which blackout occurs. During exposure to high transverse acceleration, the effects on the visual system depend largely on whether the acceleration force is $+G_x$ (eyeballs-in) or $-G_x$ (eyeballs-out). When the acceleration is $+G_x$, no major visual disturbances have been reported up to loads of $+14\ G_x$ for five seconds at peak G. At levels between +6 and $+12\ G_x$, however, there may be some tearing, apparent loss of peripheral vision, and difficulty in keeping the eyes open. For $-G_x$ (eyeballs-out acceleration), some pain may be experienced and small petechiae may occur on the lower surface of the eyelids. Vision may be temporarily impaired although, to date, no internal damage has been reported for accelerations as high as $+15\ G_x$. For $-G_x$ acceleration, however, the kind of restraints provided for the anterior surface of the body is a major consideration." (Chambers & Hitchcock, 1963).

In work done at the Naval Aerospace Medical Institute, Cochran, Gard, and Norsworthy (1954) present results based on the testing of 1,000 subjects which indicate the mean threshold for loss of peripheral vision to be 4.1 G; for blackout, 4.7 G; and for unconsciousness, 5.4 G. These thresholds are based on rates of onset of 1 G per second.

The loss of peripheral vision, occurring as it does almost at the point of blackout, represents one endpoint in what is a steadily lessening visual capacity with increasing acceleration. Figure 13-3 shows a regular decrease in foveal absolute threshold from 1 G upward. This curve indicates that at 4 G a light would have to be three times as intense as at 1 G in order to be seen.

Measures of loss of visual acuity during acceleration parallel the changes found in the absolute threshold. Figure 13-4 shows the decrease in visual acuity occurring with increase in acceleration from 1 to 8 G. At 7 G, the size of a target must be twice that at 1 G, if it is to be seen.

White (1958) describes research which studied the effects of positive acceleration on the relation between visual acuity and luminance level. Under conditions of 1 G, the expected decrease in acuity threshold with increasing luminance was found. With the application of acceleration forces of 3 G and 4 G, an increase in threshold (decrease in visual effectiveness) was found at all luminance levels, but the effect was most noticeable at low levels. At a luminance of 0.01 mL, the minimum detectable visual angle increased from 4.0 minutes at 1 G to 7.59 minutes at 4 G. At 150 mL, the change in visual angle was only 0.25 minutes between these two values of acceleration. It is apparent that the effects of acceleration forces on vision are most pronounced in the low-illumination levels occurring during night flight.

The degradation of vision under acceleration stress is primarily the result of circulatory changes within the retina. The progressive closure of the

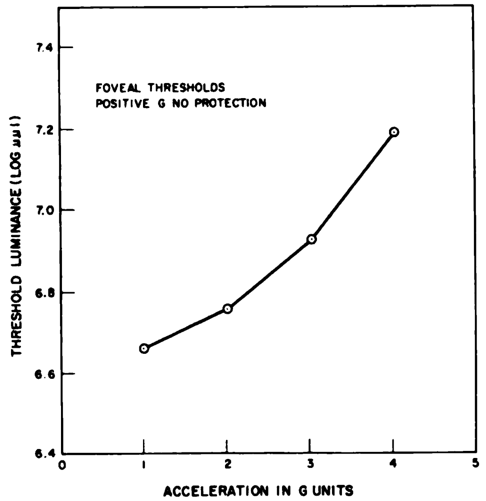

Figure 13-3. *Mean threshold luminance as a function of acceleration. One can see that acceleration of 2 G produces measurable impairment of the dark-adapted foveal threshold* (white, 1958).

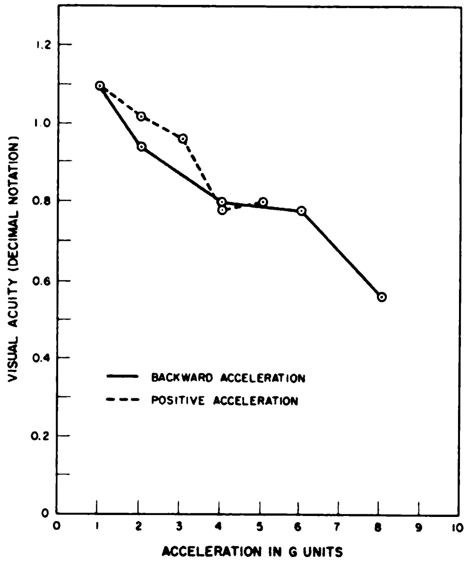

FIGURE 13-4. *Binocular visual acuity as a function of acceleration* (WHITE, 1958).

visual field experienced during exposure to G forces may be duplicated through ophthalmodynamometry (application of pressure to the eyeball). In a recent study by Jaeger, Severs, Weeks, and Duane (1964), who used a combination of ocular pressure applied by a plethysmographic (described in Weeks, Jaeger and Duane, 1964) and positive acceleration, visual symptoms were produced at or below 4 G with a rate of onset of G of approximately 1/3 G per second. By thus lowering the normal threshold of blackout by one to three G units, the physical G stress was considerably lessened and permitted the subject to concentrate on his visual changes. The visual patterns obtained showed an initial nasal dimness followed by a nasal field loss approaching close to fixation which appears almost as hemianopia. Concurrently, a peripheral temporal dimness begins, followed by concentric temporal field loss with the last remaining vision located between fixation and the blind spot.

The majority of findings concerning degradation of specific visual task performance such as visual acuity (White & Jorve, 1956), reading aircraft instruments (Brown & Lechner, 1956), and visual reaction time to discrete light signals (Brown & Burke, 1958) are associated with retinal ischemia or mechanical distortion of the physical components of the eye.

Another source of visual disturbance has been identified in relation to entopic phosphenes. Human centrifuge subjects under +G acceleration have reported impairment of vision due to "flashing lights," "fog," or "spots" before their eyes. In a study by Tobias and Meehan (1964), subjects reported seeing images described as "golden worms interspersed with cobalt blue spots" when exposed to positive acceleration levels of about 4.5 G following a slow onset rate (1.5 G per minute). These authors conclude that the acceleration phosphene underlying these visual responses is produced when reduction of blood pressure in the choreocapillaris complex permits the intraocular pressure to distend the retina and mechanically excite the first layer of interconnecting neutrons. During increased perfusion of that area, due to the carotid sinus reflex, the phosphene disappears. They speculate that pilots, during maneuvers at high G, could interpret a phosphene sensation as an external object unless there had been prior experience with entopic effects. The form and intensity of the resulting disorientation depend upon the directional and accelerative components acting on the pilot. Unfortunately, the human is incapable of differentiating the components of force to which he is exposed. He can appreciate only the effects of various forces in terms of their common resultant. When accelerative forces are combined with the normal force of gravity, which is our learned reference of verticality, such that the resultant force is at an angle to the longitudinal axis of the body, the sensation is one of being tilted in space.

There are two common types of illusions associated with vestibular stimulation which have been studied intensively in a research program at the Naval Aerospace Medical Institute under the direction of Dr. Ashton Graybiel. These are termed the "oculogyral" and "oculogravic" and are described by Graybiel (1952), Graybiel and Hupp (1946), and Whiteside, Graybiel, and Niven (1965) as follows:

Oculogyral Illusion.—This may be defined as a form of apparent motion of the visual field relative to an observer which has its genesis in the behavior of the semicircular canals on exposure to certain unusual force environments. For example, if a person, while viewing a luminous objectively stationary target in the dark, is rapidly rotated CCW, maintained at constant velocity for a minute, then suddenly stopped, he will perceive the oculogyral illusion and experience concordant sensations of rotation. Immediately with the onset of acceleration, the target appears to move rapidly and jerkily to the left followed by a loss of jerkiness with smooth gradual slowing to a stop. After a few seconds this is usually followed by a brief slow apparent motion in the opposite direction. On deceleration the events are reversed. The persistence of the illusion long beyond the accelerative stimulus rests mainly in the behavior of the cupulae-endolymph system. Ordinarily, as in turning the head, acceleration is almost immediately followed by deceleration; the cupulae, mechanically displaced by the inertia of the endolymph on acceleration, are mechanically restored to their neutral position on deceleration. In the example given above, however, there was a relatively long period separating acceleration and deceleration during which restoration of the cupulae slowly took place by virtue of their elasticity. The oculogyral illusion has different characteristics when a person is rotated about an earth-horizontal axis, the reasons for which are not entirely clear. The Coriolis oculogyral illusion results from the simultaneous rotation of the canals about two axes and has characteristics which distinguish it from the typical illusion.

The oculogyral illusion is readily perceived when an aircraft oscillates about its long axis; the viewer, although fixed in relation to the vehicle, perceives the roll as apparent movement. Any prolonged turn in an aircraft involving a period of constant velocity which permits restoration of the cupulae, will result in the oculogyral illusion, both on going into and coming out of the turn, as explained above. A good practical working knowledge of this illusion and its significance in causing vertigo in aircraft requires study and experience.

Oculogravic Illusion.—When a person is subjected to a change in direction of the gravitoinertial vertical with reference to himself, this is rightly interpreted as a tilt away from the upright, and the visual framework tends to tilt concordantly. The latter phenomenon is a form of apparent motion which has been termed the oculogravic illusion. The characteristics of the illusion include: (1) good correspondence between the rate of apparent motion and the magnitude of the apparent displacement, (2) the long delay between the actual change in the gravitoinertial vertical and awareness of the full effect of the illusion, and (3) the lack of evidence of any habituation.

The oculogravic illusion is readily perceived by an observer exposed to centripetal force on a human centrifuge if his seat is fixed with respect to the arm. Under favorable conditions, while viewing an objectively vertical luminous line in the dark, it will appear to tilt when the direction of the resultant force vector has inclined about 1.5 degrees from the earth-vertical. The observer's estimates of the tilt correspond closely to the change in direction of the resultant vector up to about 30 degrees, but beyond, the subject increasingly overestimates the angular change. Even maximal visual cues to the earth-vertical can be easily overpowered by manipulating the strength of the stimulus to receptors signaling the gravitoinertial upright;

the observer perceives tilting of the entire visual world. Under proper conditions and if the strength of the stimulus is sufficiently great, the observer feels as though he were lying on his back and perceives the centrifuge as stationary with room rotating about him. This curious reversal is probably analogous to the experience of aviators in a spin when they report that they seem to be stationary with the earth turning around them. The oculogravic illusion is most commonly perceived when aviators are exposed to linear accelerations such as on takeoff. Undoubtedly this illusion combined with the postural illusory effects has been responsible for many instances of disorientation in flight.

The fact that information provided by human sensory processes may be in complete disagreement with environmental conditions poses a serious problem for a pilot. In these circumstances, survival may depend upon ability to disregard these sensations and rely upon the instruments.

The Proprioceptive Sense

The proprioceptive sense serves as a supplement to visual and vestibular functioning. It is not at all times correct, however. For example, a reduction from a high G force (6 to 9 G) to a low G force (2 G) produces a situation in which the subject cannot recognize that he is still exposed to acceleration forces (Brown & Lechner, 1956). This is further indication of the inability of pilots to fly by "feel" alone.

Audition

The sense of hearing continues to function beyond the time when vision is lost under high acceleration. Brown and Lechner (1956) also report that auditory reaction times increase significantly with exposure to positive acceleration. They note, however, that part of the increase in response time may have been due to slower execution of the required motor response rather than in signal detection.

Subjects required to add pairs of numbers which were presented auditorily during exposure to levels of $+G_z$ up to grayout, exhibited longer response time with increasing G force (Cope & Jensen, 1961). In that experiment, the motor response required was simply to depress a button mounted on either handgrip to describe the sum as odd or even. Whether observed increases in response time in such instances are due to auditory impairment, simple distraction, or an hypoxic effect is undetermined.

THE INFLUENCE OF ACCELERATION FORCES ON BEHAVIORAL CHARACTERISTICS

Motor Performance

Any system guided by a human operator presents well-defined requirements concerning the direction, magnitude, and distance through which limb and control movements must be made. To date, no comprehensive research program has been conducted to relate the dimensions of human motor performance under acceleration stress to particular systems requirements in order to arrive at optimal design specifications. However, since the weight of the body increases in direct proportion to acceleration, it would be expected that movements required in a direction opposite to the force of acceleration require more exertion and may require more time to perform than those acting at right angles to or in the same direction as the existing gravitational forces. Brown (1961) discusses one incident in which a pilot was unable to actuate his preejection lever due to the combined loading of the lever and the forces of —G_z acceleration. The pilot had to eject through his canopy.

In cases in which an accident is associated with maneuvers producing acceleration forces, attention should be directed to specific controls which might have been rendered useless due to excessive demands on pilot motor performance.

Complex Tasks

Complex tasks presume a combination of perceptual and/or motor activities performed simultaneously or sequentially. Acceleration generally produces a decrement in performance in multicoordinate control tasks. However, work conducted by Chambers and Hitchcock (1963) has shown that well-trained test pilots are able to maintain performance above minimum requirements for a "moderately complex" tracking task involving prolonged exposure to relatively high and varied G forces such as those encountered during reentry from space.

Cognitive Processes

There is almost no evidence as to the effect of acceleration on "higher mental" abilities. Reliable and valid tests of higher mental abilities which can be administered within the basically restrictive and time-limited conditions of a high-G environment are severely lacking. In a series of experiments conducted at AMAL, involving such tasks as continuing and repetitive memorization of portions of random symbol sequences, and comparison of present to previously displayed pairs of symbols, no significant decrease in accuracy was found up to 5 G. However, impairment of "immediate memory" was found for 7 and 9 G (Chambers & Hitchcock, 1963).

In view of the sparsity of conclusive data, the problems involved in defining and testing "higher mental abilities" and the methodological obstacles to separating out sensory, attentional, and motivational factors, it would appear that specification of the effect of acceleration in this area must await further research.

PROTECTION AGAINST ACCELERATION FORCES

Under stress of positive acceleration, visual impairment and unconsciousness result from decreased arterial pressure at head level brought about by venous pooling and decreased cardiac output. The extent of these effects may be reduced by voluntary "straining" maneuvers, and mechanical application of counterpressure (antiblackout system).

Straining Maneuvers

M-1 Maneuver.—This procedure is an effective voluntary response which can reduce the threshold of blackout by about 2.4 G (Wood & Lambert, 1952). It consists of two components: (1) forced expiration through a partially closed glottis, and (2) tensing of the skeletal musculature. Bondurant (1961) provides an excellent description of the effects of the M-1 maneuver:

"The respiratory component of this maneuver consists of forced expiration against a partially closed glottis over a period of 3 to 5 seconds. This is equivalent to the scream of the early pilots. Following a quick inspiration, forced expiration can be repeated. Arterial pressure is elevated by this component of the M-1 maneuver in the following ways: As forced expiration is instituted, the resulting increase in intrathoracic pressure is transmitted directly to the aorta. There is an immediate increase of peripheral arterial pressure which is only slightly less than the increase in intrathoracic pressure. During the period of forced expiration, intrathoracic pressure falls continuously while intra-abdominal pressure remains elevated. A pressure gradient between the abdomen and the thorax is thus created which, in effect, augments the return of blood to the heart. The initial increase in arterial pressure is thus sustained throughout the period of forced expiration. This maneuver affords approximately 2.4 G protection against blackout."

Valsalva Maneuver.—This maneuver is similar to the M-1 except that instead of expiring through a *partially* closed glottis, expiration is attempted against a *fully closed* glottis. The Valsalva must be performed *before* acceleration. During the attempted expiration, arterial pressure is reduced. If acceleration occurs at that time, the blackout threshold is lowered and unconsciousness may even result. Following the maneuver, arterial pressure is elevated in response to peripheral vasoconstriction and tolerance is increased by about 1 G.

The Valsalva maneuver is less effective than the M-1 maneuver and, if not performed prior to acceleration, may be detrimental.

Pilots should be instructed on the proper execution of the M-1 maneuver and the nature of the less desirable and possibly dangerous Valsalva maneuver.

Antiblackout System

Contemporary antiblackout suits provide protection against positive radial acceleration by applying compensatory pneumatic pressure to the calves, thighs, and abdominal area.

The single pressure air-bladder suit, now generally employed, not only balances the intravascular pressures but also compresses the arteries to some extent. By increasing peripheral resistance it increases arterial pressure at heart level. The large abdominal bladder squeezes the viscera and drives blood up into the thorax. In addition, it raises the diaphragm and thus significantly decreases the heart-eye distance. The sum total of the various influences which the suit exerts is complex and results in protection both by preventing venous pooling and by increasing arterial pressure at head level. By wearing the G suit a pilot is able to withstand one to two more G's than he could without such protection.

The operational principles of the antiblackout system are illustrated in figure 13-5. A line connects the suit to a high-pressure air source on the aircraft. Air pressure to the suit is controlled automatically by a valve which is sensitive to positive (head-to-foot) acceleration. When acceleration exceeds 1.75 G, the suit is pressurized at a selected rate of 1.0 or 1.5 psi for each G above 1.75 G. In this way, the suit compresses the legs and abdomen by an amount proportional to the acceleration.

A "press-to-test" button allows the suit to be pressurized at will. On extended flights, this function is occasionally used in the absence of abnormal G forces as a means of improving poor circulation in the lower extremities associated with prolonged inactivity.

Influence of Fit on G Suit Effectiveness.—In a survey of one Navy squadron by personnel of the Naval Aerospace Research Department, it was found that over 50 percent of the flight personnel were wearing improperly fitted G suits. It was also found that the Flight Surgeon who routinely flew with the squadron was getting approximately .5 G protection out of his improperly fitted suit. Following necessary alterations, his protection was increased to 1.5 G solely through proper fitting. Flight Surgeons and parachute riggers should devote particular attention to problems of properly altering and fitting the G suit. Pilots should be monitored regularly to insure that they understand the necessity for wearing the G suit properly.

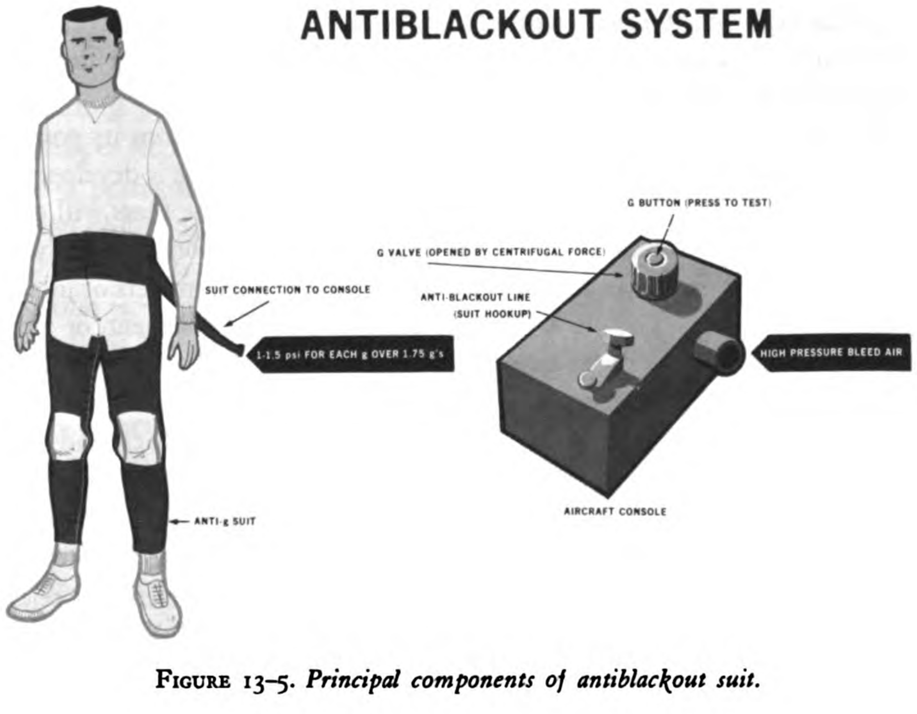
ANTIBLACKOUT SYSTEM
G BUTTON (PRESS TO TEST)
G VALVE (OPENED BY CENTRIFUGAL FORCE)
SUIT CONNECTION TO CONSOLE
ANTI-BLACKOUT LINE (SUIT HOOKUP)
1-1.5 psi FOR EACH g OVER 1.75 g's
HIGH PRESSURE BLEED AIR
ANTI-g SUIT
AIRCRAFT CONSOLE

FIGURE 13–5. *Principal components of antiblackout suit.*

VIBRATION

ALTHOUGH MORE an engineering than a medical problem, vibration has important implications for the Flight Surgeon. Vibration may affect physiological functioning, perceptual and motor performance, and personal comfort. A comprehension of the physical phenomenon of vibration is a basic requirement in the understanding of the effects of vibration on humans.

PHYSICS OF VIBRATION

A practical definition of vibration relating to human physiology has been offered by Guignard (1965), to the effect that vibration is a sustained, structure-borne disturbance, applying a translatory movement to the body and perceived by the senses other than hearing.

Any physical system which responds to displacing forces must have three essential properties: mass (or inertia), elasticity, and damping. For the present, it will be convenient to disregard the property of damping, that effect which acts as a viscous drag against the action of a displacing force, and consider the behavior of a mass supported by a spring, which represents the simplest physical system exhibiting simple harmonic or sinusoidal oscillation in response to a displacing force.

If such a system is disturbed and the mass is displaced from its position of rest or equilibrium point by a distance, x, a restoring force is developed in the spring. When the initial displacement force ceases, the mass will oscillate about its equilibrium point. This back-and-forth motion may be shown as a function of time as in figure 13–6, wherein several parameters of interest may be identified. These are (1) the magnitude of displacement, or vibration amplitude, (2) the time-rate of displacement, or velocity, (3) the rate of change of velocity, or acceleration.

As the term oscillation implies, the vibrating mass moves back and forth with reversals of direction of movement occurring at the point of maximum displacement from its resting position. For reasons which will not be considered here, it happens that the velocity of the mass is greatest as it passes through its equilibrium point, and the rate of change of velocity (acceleration) is greatest at the point of maximum displacement or "peak amplitude."

A mass is said to have completed one "cycle" when it has left its resting

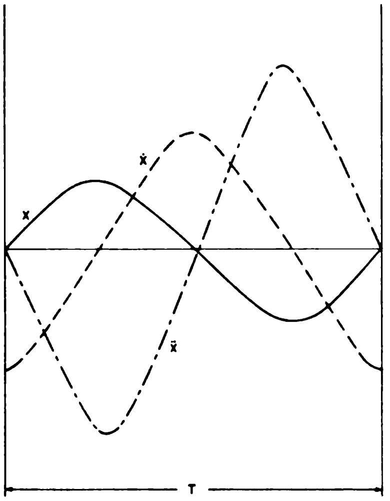

FIGURE 13-6. *Diagram showing time relationships of displacement* (x), *velocity* ($\dot{x}$), *and acceleration* ($\ddot{x}$) *for sinusoidal vibration. T is the period of the vibration: 1/T is the frequency.* (GUIGNARD, 1965 BY PERMISSION OF THE CONTROLLER OF HER BRITANNIC MAJESTY'S STATIONERY OFFICE.)

position, moved to peak amplitude in one direction, reversed direction and traveled to peak amplitude in the opposite direction, and then returned to its starting position. The "frequency" of vibration is usually specified as "cycles per second" (cps).

As a practical matter, the effects of sinusoidal or periodic vibration on human physiological functioning and performance are primarily a function of the acceleration component of vibration. The "intensity" or severity of vibration is frequently described in terms of its acceleration component, e.g., centimeters per second per second $(\mathrm{cm/sec})^2$.

A familiar quantity is the rate of change of velocity imparted to a free-falling object by the force of gravity. This quantity is termed the gravitational constant (G) and is equal to approximately 980 $\mathrm{cm/(sec)^2}$ [i.e., $G = 980\ \mathrm{cm/(sec)^2}$].

Thus any acceleration given in $\mathrm{cm/(sec)^2}$ may be expressed in units of G by simply dividing the quantity by 980 $\mathrm{cm/(sec)^2}$. For example, a vibration whose peak acceleration is 196 $\mathrm{cm/(sec)^2}$ could be expressed equivalently as: $[196\ \mathrm{cm/(sec)^2}]/[980\ \mathrm{cm/(sec)^2}] = 0.2$ G.

PARAMETERS OF VIBRATION

A number of factors other than those inherent within vibration itself partially determine the effects of vibration. The effects of vibration depend upon the duration of exposure. A related factor is the schedule of exposure—continuous or intermittent. Perhaps a more relevant question than, "How long can an individual tolerate vibration?" is, "How long can an individual tolerate extended exposure to intermittent, short-time vibration?"

Vibration may be applied to the body in a standing, sitting, supine, or semisupine position. An individual reacts differentially to vibrations as a function of body position. In addition to the orientation of the body, the direction of vibration with respect to the body and with respect to gravity is also important. A great deal of confusion regarding direction of vibration and orientation of the body has been eliminated by adopting the terminology—outlined in the acceleration section of this chapter—used to describe acceleration forces. The type of vibration known as complex or random is characterized by varying frequencies and amplitudes appearing at intermittent intervals. Body movements can correspond to pure sinusoidal or complex vibration, which may occur in any or all of three axes.

Performance during vibration is affected by the differential vibration between an individual and the task to be performed. If the individual is vibrating while the task is not, or vice versa, performance differs from that when both individual and task are vibrating together. Important, but often neglected, aspects of vibration are the effects on reaction to vibration of motivation, learning, or adaptation during vibrations. For instance, it is a well-known fact that pilots intent upon controlling an aircraft during a difficult maneuver can tolerate buffeting without ill effects, while crewmen or passengers may suffer from motion sickness. An understanding of the effects of vibration in operational aviation is made even more obscure when the possibility of interactions between vibration and other environmental and psychological stressors is considered. There is very little experimental evidence concerning the effects of *vibration in combination with other stressors,* yet vibration rarely occurs isolated from other stressors (Hornick, 1962a).

VIBRATIONS IN AIRCRAFT

The medium through which vibration acts upon an aviator is the aircraft. Many of the vibration problems associated with early aircraft have been substantially reduced with engineering advances. For instance, "snaking" (yaw oscillation) and "Dutch roll" (yaw oscillation accompanied by roll oscillation) have been reduced considerably in many aircraft by the inclusion of an electronic yaw damper. However, the problem of vibration in aircraft is by no means entirely solved. Engineering advances which have made high-performance aircraft possible introduce new vibration problems.

Vibration is a result of (1) the propulsion system of the aircraft, and (2) the structure of the aircraft, as they interact with the supporting air. Although jet aircraft have minimal vibration originating in the engine, their increased performance capabilities introduce unique structural and aerodynamic vibration. The dynamic pressure of the air on the aircraft varies with gusts, terrain turbulence, speed, altitude, and maneuvers. Generally, different vibrations can be expected with different types of aircraft. Reciprocating-engine, helicopter, turbojet, turboprop, and rocket aircraft exhibit unique vibrations. In addition, different aircraft within these classes may have distinct vibration characteristics. The vibration associated with these aircraft also varies with flying conditions and maneuvers. Vibration may be continuous or intermittent, barely perceptible or extremely annoying.

Fraser (1964) determined the vibration levels in a T-33 aircraft during low-altitude, high-speed (LAHS) flights. Although vibration was complex, the average frequency for this aircraft was 2.35 cps and varied up to 4 cps. Acceleration, measured at the head, varied up to 4 G. Impacts of 0.5 interspersed with peaks of 1 G or greater were common. In the most severe flight, accelerations of 2 G or greater occurred on an average every five seconds for more than 35 minutes. Jolt was relatively low—from 10 to 40 G per second and occasionally over 100 G per second. These determinations are probably similar to what can be expected in most LAHS missions. Generally, high-performance jets in LAHS missions, and rockets during launch and reentry, exhibit low-frequency (below 20 cps), high-amplitude vibrations for periods lasting only seconds or fractions of a second. Propeller-driven planes, on the other hand, exhibit high-frequency (above 20 cps), low-amplitude, continuous vibrations which originate primarily in the engine(s).

Due to various damping devices and the transmission characteristics of the human body, the relationship between the vibration of an airman and the vibration of the aircraft may be quite complex. The aircraft, parts of the aircraft (such as instruments and controls), the whole body or parts of the body (such as arms, legs, or head) may all exhibit the same or different vibration characteristics. Each of these diverse vibrations poses distinct problems.

EFFECTS OF VIBRATION ON HUMANS

A number of controlled laboratory experiments have been performed to determine the effects of vibration on humans. These studies have investigated physiological effects, subjective tolerance levels, mechanical body responses, and various aspects of human performance as they relate to frequency and acceleration (or amplitude) of vibration as a function of various parameters.

Continuous vibrations above 20 cps in aircraft are annoying but generally are not harmful. Exposure to these frequencies during extended missions may cause discomfort and fatigue.[1] Between one and 20 cps, vibrations can cause physiological damage and hinder performance. Below one cps, waves with high amplitude and high accelerations generally cause "motion sickness." Most laboratory experiments have emphasized the vibrations in the range from 1 to 20 cps because (1) these are the vibrations which occur most frequently in modern aviation, and (2) the effects of these vibrations are most detrimental to flying. Unfortunately, these experiments do not answer, or only partially answer, many of the crucial questions concerning the effects of vibration on humans.

Impedance Studies

Impedance studies attempt to explain physiological changes, subjective tolerance levels, and performance under vibration in terms of the dynamic characteristics of the human body and parts of the body. These characteristics are quantifiable and can be used to describe the mechanical transmission characteristics of the body or parts of the body, to estimate vibration at various parts of the body, and to estimate the utility of various protective devices. The approach involves the assumption that the body is a linear, passive mechanical system. The assumption that the body is passive is convenient but oversimplified. At many frequencies and amplitudes the body is not passive. In addition, tissue responses are frequently nonlinear. The approximations of body or part-body responses which are possible if these assumptions are accepted, however, can be quite useful.

For low-frequency vibrations, it is possible to describe human responses by lumping systems of the body and describing them in terms of mechanical circuits. Figure 13-7 presents a mechanical analog of the human body. The head, for example, is represented by a mass suspended on a spring with frictional resistance. Other body subsystems are not so simply depicted as the head. Impedance studies attempt to determine the appropriate values for the components of mechanical circuits by measuring impedance. Mechanical impedance, in analogy to electrical impedance, is defined as the ratio of the transmitted *force* to the *velocity* at the point where the force is applied. With this information, it is possible to estimate the absolute mechanical energy transmitted to the body (or body subsystems).

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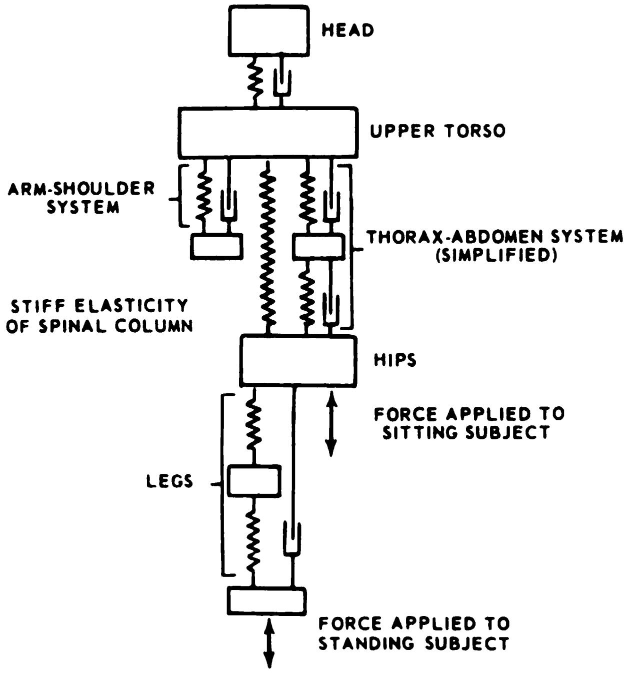
[1] It is possible for an individual to be uncomfortable due to vibration, yet to perform a task at a satisfactory level. The reverse, of course, is also true.

Impedance studies have given valuable information concerning (1) the body resonances at 4 to 6, and 10 to 14 cps, (2) head resonance at 20 to 40 cps, (3) thorax-abdomen resonance at 3 to 4 cps. They have predicted the differential physiological effects due to body position and restraint systems. Further information on impedance studies is available in Coermann (1962), Coermann, Ziegenruecker, Wittwer, Von Gierke (1960), and Goldman and Von Gierke (1960).


HEAD
UPPER TORSO
ARM-SHOULDER SYSTEM
THORAX-ABDOMEN SYSTEM (SIMPLIFIED)
STIFF ELASTICITY OF SPINAL COLUMN
HIPS
FORCE APPLIED TO SITTING SUBJECT
LEGS
FORCE APPLIED TO STANDING SUBJECT

This simplified model of the body as a system of masses, springs, and dampers was constructed from impedance data and other measurements of displacements of individual organs under vibration. Mechanical characteristics of body masses may be so represented for a specified frequency range.

FIGURE 13-7. *Mechanical analog of the human body* (WEBB, 1964; SOURCE: COERMANN ET AL.).

Subjective Tolerance Levels

Subjective tolerance refers to the degree of vibratory stress individuals are willing to undergo voluntarily. Subjective tolerance levels have been investigated primarily to specify acceptable levels of vibration in the structural design of aircraft. They are of interest to the Flight Surgeon because, in spite of structural requirements, vibration levels may occasionally approach the subjective tolerance levels of individuals. One of the most noteworthy findings of tolerance-level experiments is the wide variation of individual reactions to vibration (Harris & Shoenberger, 1964). Another problem is that different studies of tolerance levels have produced discrepant results. In spite of these deficiencies, a number of generalizations are possible. The tolerance curves shown in figure 13–8 serve as general indications of the levels of vibration which are tolerable in operational aviation.

Figure 13–8 presents the results of several tolerance studies. The first curve (Temple, Clarke, Brinkley, & Mandel, 1964) presents the tolerance to vibration while supine, restrained on a contoured couch, with vibration and gravity acting for short times in the x-axis. The second curve (Magid, Coermann, & Ziegenruecker, 1960) presents the tolerance level for subjects seated in a T–33 aircraft seat, restrained with lap belt and shoulder harness, with gravity and vibration acting for short times, less than one minute, in the z-axis. The third curve (Mandel & Lowry, 1962) presents the tolerance level for seated subjects, restrained with lap belt and shoulder harness, and with vibration and gravity acting for one minute in the z-axis. Curve 4 (Magid et al., 1960) presents one-minute tolerance for the same conditions as curve 2. The fifth curve (Goldman & Von Gierke, 1960) presents averages of a number of studies for exposure times ranging from five to twenty minutes for the standing, sitting, and lying positions with no protection. The sixth curve is the WADC long-time acceptable vibration exposure level for military aircraft (Getline, 1955).

All of the tolerance level curves exhibit a similar relationship between tolerance and frequency. Minimum tolerance is at 6 to 8 cps for all of the curves. This low tolerance level occurs because the body has a natural resonance between 4 to 8 cps. This means that the body, if displaced from the resting position and then released, vibrates between 4 and 8 cps. Vibration to the body between 4 and 8 cps is amplified by the natural resonance of the body. This amplification may cause accelerations of the head to be as much as three times as great as those at the seat (see Coermann et al., 1960).

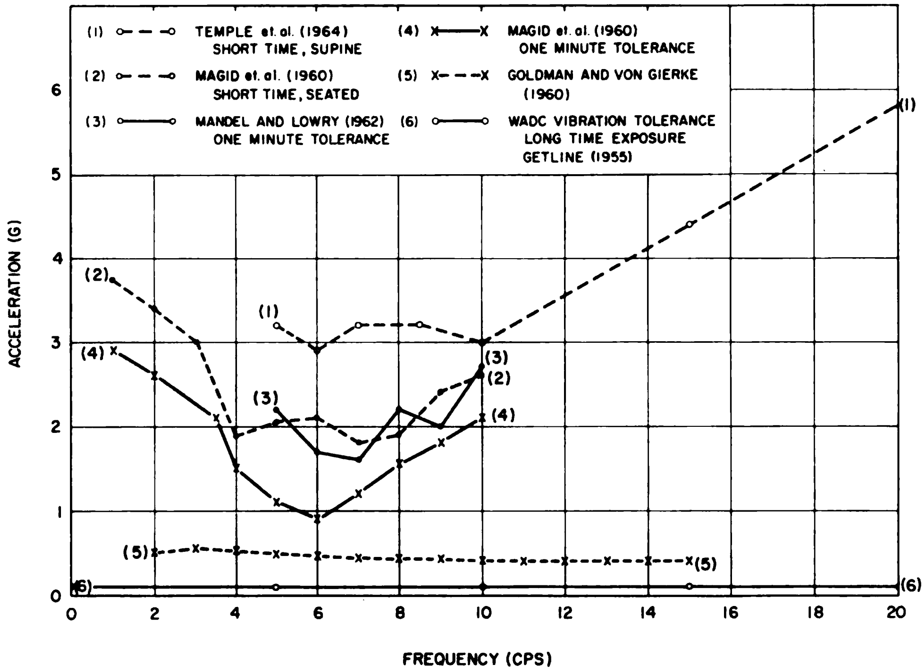


FIGURE 13-8. *Levels of tolerable vibration in operational aircraft.*

It is clear also that man's ability to tolerate vibration rises rapidly as frequency increases or decreases from the 4 to 8 cps range.

The first curve of figure 13-8 presents the conditions and tolerance levels which are associated with manned spacecraft during launch and reentry. Vibration exposure is very brief. Restraints and supine position make it possible to tolerate intense vibration. The second curve indicates man's ability to tolerate the type of vibration associated with LAHS flight using the type of restraint system found in Navy aircraft. It is unlikely that vibration as severe as that indicated as intolerable by curves 3 and 4 is experienced by aviators for as long a period as one minute. Curves 3 and 4 are the only curves which represent the same vibration conditions. Although they should be the same, discrepancies exist between the two.

Many short, intermittent vibrations for extended intervals are associated with LAHS flight. It has not been determined at present how individuals tolerate the vibrations shown in curves 1 to 4 when these are repeated at random intervals for an extended time.

Motivation and other psychological factors undoubtedly determine how much discomfort and pain an individual will tolerate as he approaches the damage level, although damage to tissue-organ systems determines the absolute tolerance level. A relationship between body size or physical condition and tolerance level has not been demonstrated (Mandel & Lowry, 1962), although commonsense—and some theoretical work—suggest an inverse relationship. Tolerable vibration may result in fatigue or in a decrement to performance. It is quite probable that low levels of vibration contribute to fatigue, but the precise fatigue effects of vibration are difficult to determine because of the elusive nature of the concept of fatigue.

EFFECTS OF VIBRATION ON PERFORMANCE

The capability of the aviator to perform the duties of flying while exposed to vibration are of paramount concern in aviation safety. Performance may deteriorate for two reasons. First, the channels for obtaining information may be affected by vibration, causing an indirect interference with performance. Secondly, the actual control actions may be directly changed by vibration. If the information necessary for flying is reduced, removed, or distorted, performance may deteriorate. If the information is available, but the aviator is physically incapable of performing the necessary tasks with the required degree of precision, performance may also deteriorate. Studies of the effects of vibration on performance have investigated these two means of interference with performance.

Information Channels

The visual sense is the primary channel for information in aviation but auditory, kinesthetic, and vestibular senses are also important.

Visual acuity is adversely affected by vibrations at frequencies above approximately 4 cps. Although the evidence is inconsistent, it appears as if visual acuity is progressively impaired as frequency increases from 4 cps to 12–30 cps, and then remains relatively constant to 90 cps, with slight decrements at 40–70 cps (Hornick, 1962a; Lange & Coermann, 1962; Mozell & White, 1958). Visual acuity is also impaired as amplitude or acceleration increase.

There are indications (see Lange & Coermann, 1962) that natural resonances of the head and of the eyeballs may contribute to visual impairment. In a T-33 aircraft (Fraser, 1964), accelerations at the head were 20 percent higher than at the seat. Vision under these conditions was poor. Damping, which causes aviator vibrations and instrument vibrations to be different, may also increase visual impairment.

It is doubtful that vibration itself interferes with *audition*. The distinction between vibration and noise is, however, an arbitrary one. Above 20 cps, noise and vibration may be produced by the same compression waves. The main distinction between the two is the medium through which they travel. Rarely in operational situations, are vibrations above 20 cps found without noise. Vibration above 20 cps will not ordinarily interfere with audition although the noises which are commonly associated with vibration may. Speech is difficult under severe vibration. The inability to speak in the normal manner at low frequencies may disrupt communication.

Touch (*cutaneous*) *senses* and the *kinesthetic senses,* which report activity of body muscles, are undoubtedly affected by vibration. Because of the nature of these senses, it has not been determined how they relate to performance under vibration. These senses invariably work in conjunction with other senses, particularly the vestibular and visual senses, and the effect of vibration on them has not been isolated.

The *vestibular senses* provide information concerning the orientation of the body with respect to gravity and accelerations of the body. Coermann, Magid, and Lange (1962) found that blindfolded subjects, attempting to maintain an upright position in an equilibrium chair while vibrating, exhibited poorer performance between 3 and 12 cps than when free of vibration. At frequencies above 15 cps, there is an indication that the ability to maintain equilibrium is actually improved under vibration. Since the subjects were blindfolded, information concerning the orientation of the chair was provided only through the vestibular, kinesthetic, and cutaneous senses. One possible explanation for the decrement and increment in performance is that the input through these channels was differentially affected by vibration at different frequencies. The changes in performance may also have been due to inability to perform the task with precision when vibrated. Excitation of the vestibular senses during high-amplitude vibration at one cps or less frequently results in motion sickness.

Tracking

A limited number of studies using various methodologies and diverse tracking tasks have attempted to determine how vibration affects tracking performance. They have attempted to identify frequencies, amplitudes, or accelerations at which tracking performance is impaired. These studies have not been successful in this respect, since performance apparently does not deteriorate as a function simply of frequency (or amplitude or acceleration). It seems probable that at one frequency-amplitude combination, a particular amplitude may be the most important factor in performance decrement while at other frequency-amplitude combinations, either frequency or amplitude may be the most important factor.

Because of the many unanswered questions in vibration research, about all that can be said concerning vibration and tracking performance is that between approximately 1 and 20–30 cps, tracking performance impairment is related, in an undetermined way, to amplitude, acceleration, and frequency. The greatest decrement to performance appears between 4 and 8 cps, the extent of the decrement, depending, of course, on amplitude.

Performance also may deteriorate above 10 cps when visual acuity is important in task performance. There are indications that vibration in the z- and y-axes hinders performance more than vibration in the y-axis (Fraser, Hoover, & Ashe, 1961), although the effect of vibration in more than one axis at once has not been determined. As vibration gets closer to the tolerance level, performance gets progressively worse (Chaney & Parks, 1964). There is also evidence that very low levels of vibration can interfere with performance but for a different reason. When vibration is severe, an extra effort is made to control the task, while a barely perceptible level of vibration is often ignored, allowing performance to deteriorate.

PATHOLOGY

There are indications that the random vibration found in aviation, which is well within tolerance limits, may result in chronic damage over a period of time. Studies of truck and tractor drivers have shown that osteoarthritis, traumatic fibrositis, herniated discs, coccygodynia, and lumbosacral pain (Magid & Coermann, 1963) may be found in persons subjected to tolerable vibrations of long duration. Pilots who regularly fly LAHS missions under heavy buffeting, and helicopter pilots, in particular, may be exposed to similar tolerable vibrations which could cause chronic damage over time.

Vibration above the tolerance level can result in physiological damage to a number of body organs. Generally, according to Magid et al. (1960), those organs having relatively large mobility and low resonance frequencies are centrally located and produce sensations at lower frequencies; conversely, those organs having relatively little mobility and higher resonance frequencies are peripherally located and produce sensations at the high frequencies. This demonstrates that the sensations are to some extent frequency dependent and that at least one etiological factor responsible for subjective response is the resonance of organ systems or subsystems.

These symptoms were observed at amplitudes (or accelerations) which were close to subjective tolerance levels. Although Magid and Coermann (1960) chose to emphasize the frequencies at which these symptoms occurred (see figure 13-9), it must be remembered that the symptom is also a function of amplitude of vibration. The following is Magid and Coermann's (1963)[2] attempt to determine etiology of the various symptoms.

Abdominal Pain.—It is believed to be caused by stretching and deformation of the terminal ileum, cecum, hepatic flexure and transverse colon and of their supporting mesenteries.

Chest Pain.—Chest pain most commonly occurs between 5 and 9 cps. Displacement resulting in stretching of the major vessels originating at the base of the heart including the aortic arch is a probable source of pain. Also, mechanical stimulation by the vibrating heart on the diaphragmatic pericardium and the parietal pericardium about the base of the heart is another possible source of pain. Displacement of the diaphragm at its anterior attachments could produce chest pain, but this would be expected to be experienced about the sternum radiating outward bilaterally.

Testicular Pain.—Here pain is evoked by the alternating displacements of the spermatic cord and possibly deformation of the testicles and surrounding tissues. The displacement necessary to elicit intense pain in these structures is very small because of their pain fiber distribution and characteristics.

Head Symptoms.—These are more difficult to evaluate. Part of the sensation is probably due to displacement of facial skin and subcutaneous tissues about the underlying bony structures. The role of the brain, spinal cord, cere-

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[2] From "Human Response to Vibration" by E. B. Magid and R. R. Coermann. In *Human Factors in Technology*, E. Bennett, J. Degan, and J. Spiegel (Eds.), copyright 1963, McGraw-Hill. Used by permission of McGraw-Hill Book Company.

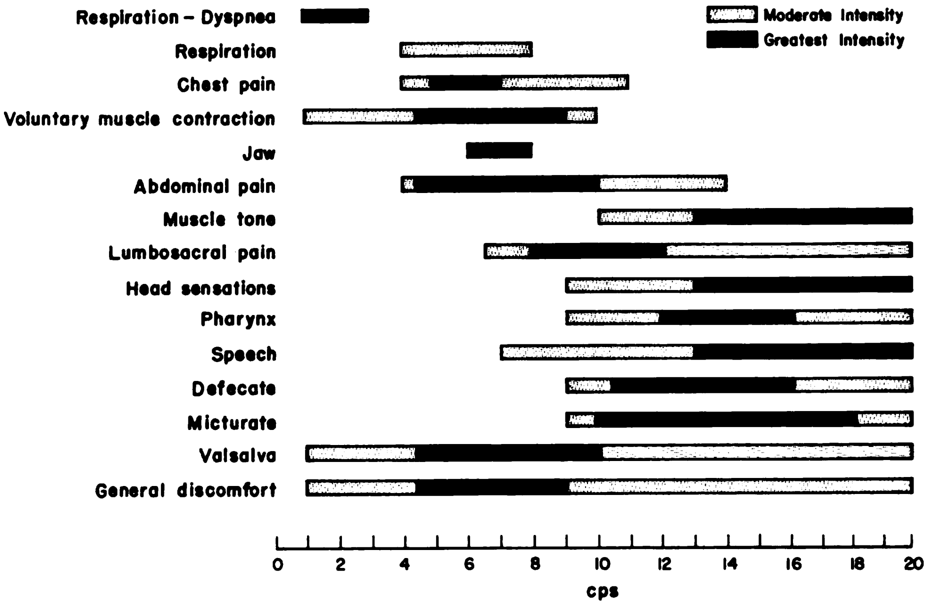

FIGURE 13–9. *Range of perceptible sensations.* (AFTER E. B. MAGID AND R. R. COERMANN, "THE RECTION OF THE HUMAN BODY TO EXTREME VIBRATIONS." Proc. Inst. Environmental Sci., 1960, P. 135. FROM "HUMAN RESPONSE TO VIBRATION" BY E. B. MAGID & R. R. COERMANN, IN HUMAN FACTORS IN TECHNOLOGY, E. BENNETT, J. DEGAN, & J. SPIEGEL (EDS.), COPYRIGHT 1963, MC GRAW-HILL. USED BY PERMISSION OF THE INSTITUTE OF ENVIRONMENTAL SCIENCES AND THE MC GRAW-HILL BOOK COMPANY.)

bralspinal fluid and vasculature in the origin of the symptomatology must still be defined. In some studies, results are confusing due to a lack of head restraint. During vibration, the unrestrained head rapidly gets out of phase with the torso and begins impacting the table. Reports of headache, facial pain, and other symptomatology resulting from such impacts are *not* limiting factors to vibration. Such limiting factors for the head must be determined with the best possible restraint or "coupling" of the head to the remainder of the body.

Dyspnea.—This occurs between 1 and 4 cps and is the result of alternating displacements of the thoracoabdominal system. Physiological respiratory movements are interfered with by the superimposed oscillating movements of the vibrating platform acting on the mass of the abdominal contents and the thoracic cage. Other possible factors contributing to dyspnea are possible changes in pulmonary hemodynamics such as pooling of blood in the pulmonary vessels resulting in pulmonary congestion.

Anxiety.—Anxiety is caused by a multiplicity of reasons among which pain, stimulation of the proprioceptive system, and respiratory impairment play important roles.

General Discomfort.—This refers to an overall subjective response. Pain, respiratory difficulties, the sensations of rapid displacements of voluntary muscle groups and extremities, including large joints and viscera, all are included in producing a general impression of discomfort.

Jaw.—The mandible and its surrounding soft tissues apparently resonate between 6 and 8 cps.

Pharynx.—The sensation of the pharnyx is described as a "tug" or "lump" in the throat quite similar to "globus hystericus." Apparently, resonance of the trachea and possibly main stem bronchi occurs at these frequencies and the sensations are the result of the trachea mechanically stimulating the sensory receptors of the supporting pharyngeal tissues.

Speech.—At the lower frequencies speech is secondarily affected by the mechanical reactions of the thoracoabdominal system. At the higher frequencies the superimposed transmitted vibrations to the trachea, particularly at the laryngeal area and possibly to the main stem bronchi, have a greater detrimental effect upon phonation.

Pelvis.—Between 10 and 18 cps, individuals experience a severe urge to micturate, and between 10.5 and 16 cps, a severe urge to defecate. Following an exposure, the urgencies immediately subside.

Voluntary Muscular Contraction.—Voluntary muscular contraction reaches maximum intensity between 4.5 and 9 cps. At these frequencies the subject has a tendency to bounce with relation to the seat regardless of the security of the lap belt and shoulder straps. The abdomen, thorax, and extremities are alternately forcibly displaced.

Muscle Tone.—Between 13 and 20 cps, individuals experience an involuntary "tightening" sensation of the lower extremities, back, neck, and head. It is suggested that at these accelerations, the transmitted force and perhaps resonances of the skeletal musculature increase the mechanical stimulation of the myotatic receptors, thereby increasing muscle tonus.

Lumbosacral Pain.—Lumbosacral pain is most intense between 8 and 12 cps. The pain is moderate to severe, occurring at the lumbosacral area, and is probably the result of exaggeration of the physiological lordotic configuration of the region and possibly compression of the intervertebral tissues. The pain subsides immediately following cessation of vibration.

Valsalva Maneuver.—A partial or complete Valsalva maneuver is performed with reflexlike characteristics and is of greatest intensity between 4.5 and 10 cps. This coincides with the frequencies having the greatest effect upon the abdomen and thorax. This maneuver increases positive pressure within the thorax, which is transmitted to the abdominal compartments. This has the effect of stiffening the walls of these compartments thereby compressing the viscera. By performing this maneuver, displacement of the viscera is minimized and protection is afforded against the alternating forces.

Respiration.—Individuals state that the superimposed passive oscillations of the thoracic cage and diaphragm upon the physiological respiratory movements interfere most between 4 and 8 cps. This sensation does not include dyspnea.

When vibration ceases, individuals may experience facial flush and, following this, some dysphoria and euphoria. Two to four hours later, most individuals experience weariness and for several hours become somewhat depressed. These findings suggest alterations in metabolic and hormonal secretions during and following severe vibrations.

These above are the symptoms which are experienced while seated in an aircraft seat and restrained with lap belt and shoulder harness with near-tolerance-level vibration acting for as long as three minutes in the z-axis. For a more thorough discussion of these symptoms, see Magid and Coermann (1963). Symptoms are slightly different if the body position is changed or if vibration is applied in a different axis. Temple et al. (1964) discuss the symptoms associated with vibrations experienced in the x-, y-, and z-axes while supine.

THERAPY

The Flight Surgeon must be aware of the problems of severe vibration. If an airman complains of symptoms which could be the result of vibration, he should be given an examination oriented around these symptoms. Treatment should be symptomatic, but attention should be directed toward prophylaxis of future symptomatology by use of whatever means available.

Protection From Vibration

Any protective device or system must, in addition to reducing the effects of vibration, be compatible with proper pilot functioning and with other protective systems. If a vibration protection system degrades another protective system (such as the ejection system) or a component of performance (such as communication or vision), it is not acceptable.

Cushions and Springs.—It is possible, in principle, to decrease the vibration received by man in an aircraft by placing a cushion or spring of the correct resiliency between him and the aircraft, perhaps with a vibration damper. In severe vibration, such protection is of limited value, since even if the man were perfectly motionless, the aircraft, controls, and instruments would still vibrate. Performance in such a situation is still adversely affected. Before using a cushion, consideration must be given to the damage which could result from a cushion or spring should ejection become necessary.

Rigid Restraints.—Plaster casts around the stomach are effective in reducing physiological damage to animals at high levels of vibration. Physiological damage generally occurs because of the displacement of inner organ systems at resonance. The casts reduce damage by preventing displacement of the organs. It is conceivable that a rigid "girdle" which could be removed and replaced at will might make it possible for astronauts to tolerate more severe vibration during the blastoff and reentry phases of space flight. Such a girdle is of little value during conventional missions, since the severe vibrations which cause organs within the chest and abdomen to displace rarely occur in aircraft (Cope, 1959). In all probability, such severe vibration results in loss of control of the aircraft well before the physiological tolerance level is reached. The additional problem of discomfort from such girdles during extended missions must also be considered.

Pressure Suits.—A natural extension of the plaster cast approach to protection is the use of pressure suits in giving added support. Coermann (1962) found that subjects wearing an MC3 pressure suit at 5 psi pressure and sitting unrestrained, had a lower tolerance to vibration than without the pressure suit. Coermann considered this reduction in tolerance level to be a result of the addition of elasticity in the form of pressure tubes and bladder, which resulted in added resonance of the body. Whether or not an inelastic pressure suit used with a good restraint system is helpful in controlling vibration has not been determined.

Restraint Systems.—A proper restraint system can also help reduce the effects of vibration during conventional flight. A lap belt improves performance during vibration by preventing the frictional rubbing and "banging" which result when individual and seat become out of phase. A tight shoulder harness, as long as it does not hinder proper respiration, can prevent many of the deleterious effects of vibration.

The value of a head restraint system in reducing vibration effects has been demonstrated for space flight (Temple et al., 1964). It appears that a head restraint system may be a valuable adjunct to a lap belt and shoulder harness at frequencies near the natural resonance of the body, but this has not been demonstrated at the present time.

Although a valid and systematic investigation of the effectiveness of various restraint systems in reducing vibration symptoms has not been made at present, Von Gierke (1959) has indicated the usefulness of proper restraint in the vibration environment. The value of proper restraint for space flight is clear. Temple et al. (1964) have demonstrated that supine individuals securely restrained on contoured and adjustable couches can tolerate a considerable level of vibration (see figure 13-8).

The Legs.—The legs, when it is possible to stand while being vibrated, are known to absorb vibration. Standing with legs bent at the knees helps attenuate vibration at the upper body and head by isolating vibration forces to the legs. This is the reason tractor drivers frequently work standing up. Hornick (1962b) found that, although legs attenuate vibration considerably at first, their effectiveness decreases slightly within two minutes. This decrease in attenuation is probably due to tiring of the leg muscles.

Respiration.—Mandel and Lowry (1962) have suggested the importance of regular respiration during vibration. They feel that a regular pattern of breathing can increase tolerance to vibration by reducing the aerophagia associated with erratic respiration. Many pilots may tense muscles and hold their breath during severe vibration. They should be made aware of the value of relaxation and proper breathing in reducing adverse vibration effects.

Selection.—In view of the vast and consistent individual differences in ability to tolerate vibration, Harris and Shoenberger (1964) have suggested that individuals be selected for missions where severe vibration is expected on the basis of their ability to tolerate vibation. Although this unique approach to the reduction of vibration problems is probably not practical at this time, it may hold promise for the future.

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CHAPTER 14

Emergency Escape From Aircraft

- GENERAL
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- BAILOUT
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  - Parachute Landing
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- SPECIAL ESCAPE PROBLEMS
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GENERAL

THE REQUIREMENT for escape of personnel from aircraft may arise at any time. When such a moment occurs, equipment for escape and survival must be available and must have been properly inspected prior to flight. Contingency plans must have been made, training completed, and aircraft equipment thoroughly checked. No time remains for last-minute preparation.

Even though escape procedures and equipment are so obviously important, aviators occasionally treat the parachute, the ejection seat, the oxygen regulator, and the crash helmet as if designed for use only by others. The Flight Surgeon can greatly assist the cause of flight safety by emphasizing various aspects of emergency escape during lectures to groups of aviators. The Flight Surgeon may discuss such topics as acceleration forces to be expected during escape, and the effects of these forces; underwater escape and the use of oxygen breathing equipment underwater; and the importance of correct positioning procedures prior to ejection. He may also serve by reminding aviators to check their personal equipment and aircraft equipment before boarding, and by calling attention to the training required by NATOPS manuals and other pertinent directives, particularly OPNAV Instruction 3740.3 series, BUMED Instruction 3740.1 series, and OPNAV Instruction 3710.7 series (the NATOPS manual).

Psychological Factors of Escape

Escape is always preceded by a decision to escape, and a delayed decision may be fatal. Psychological factors contributing to delay include:

1. Fear of the unknown.
2. Attempt to salvage aircraft.
3. Fear of career jeopardy.
4. Ignorance of precise limits of ejection seat performance.

Accident experience indicates that a large percentage of fatal accidents occur at altitudes where survival is virtually assured, yet delay in making the escape decision has apparently been a major factor. The underlying causes for decision delay can only be surmised.

The pilot considers his aircraft a friendly, known environment, but the escape environment represents the unknown. Unless his aircraft environment changes drastically (uncontrollable fire, structural failure, or the like), it is reasonable to assume he feels considerable reluctance to exit the cockpit.

Interviews with pilots who have successfully escaped from aircraft reveal there is a considerable feeling that every attempt should be made to "bring the aircraft back." This is evident even in cases in which there was no conceivable way to save the aircraft. A number of factors may condition this attitude. Most pilots know their aircraft are extremely expensive, and that any accident will be thoroughly scrutinized by investigating personnel. This may lead the aviator to fear that his career will be jeopardized. He may feel that some unintentional or inappropriate procedural error placed him in the emergency situation or that his emergency procedures (corrective action) missed some vital system element and that by repeating the procedures his aircraft and his professional pride can be saved.

Ejection seat systems have greatly improved in recent years, particularly in the low-altitude, low-airspeed regimes. This also may contribute to decision delay by giving the pilot more time to attempt remedial action, causing him possibly to exceed safe escape parameters.

It is imperative that pilots know the escape parameters (as covered in NATOPS) for their particular aircraft. The basic requirement is that the proper mental conditioning or "set" be established so that the escape decision can, and will, be made while escape is still possible.

The Parachute

All current escape systems utilize a parachute to lower escapees to the surface at a safe vertical velocity. In referring to parachutes, the alphanumeric designation of the assembly is used, such as NB-7. The pack type (back, seat, or chest) may be referred to. The canopy size and shape may also be mentioned in terms of diameter of the canopy skirt: 28-foot, 24-foot, or 26-foot conical. Personnel parachutes consist of three primary parts: canopy, pack, and harness.

The canopy typically is a fabric hemisphere made up of a series of triangular panels called gores which radiate from an opening (vent) located at the apex of the hemisphere. The panels terminate at the circumference of the hemisphere (skirt). Modern parachutes are constructed from nylon fabric which has been woven in a manner which provides resistance to "running" (rip-stop nylon). The elasticity of nylon absorbs some of the energy during initial opening or inflation of the canopy.

Nylon suspension lines (shroud lines) are sewn into the canopy and are continuous. They extend from one or two confluence points, which are the points where nylon webbing connects the parachute to the harness assembly, up through the seams connecting each triangular panel, and back to the opposite confluence point. Again the nylon used in suspension line construction aids in absorbing energy during opening. Nylon webbing (risers) attached to connector links at the confluence points provides the attachment between the parachute canopy and the harness worn by the aviator.

The pack maintains the parachute in a ready state, protecting the canopy and suspension lines during normal flight operations and handling and protecting the parachute from the effects of windblast which could cause premature deployment. Pack design and the method of packing the canopy and suspension lines within the pack provide an optimum, predictable, and orderly deployment of the assembly when actuated.

The harness typically consists of a series of nylon web straps designed to contain the body properly and to distribute opening forces. The design of parachute/harness assemblies differs in relation to the performance characteristics of the aircraft, the amount of space available in the seat or aircraft, and the ejection system employed. In some aircraft the occupant wears a harness as part of his basic flight clothing and once seated, attaches the parachute pack. In others, the crewmember dons a harness which permits him to move about the aircraft in the conduct of his normal flight duties. When an emergency arises, a parachute pack is obtained from a storage location and is attached to the harness. In still others, the harness is an integral part of the parachute and the entire assembly is worn throughout the flight.

All personnel parachutes have a smaller parachute (the pilot or drogue chute) attached to the apex of the main parachute canopy. It deploys first, is forcibly opened by a wire spring assembly, and extracts the main canopy, thus assuring rapid, predictable deployment of the main canopy and causing the suspension lines to be partially tensioned prior to the application of main canopy opening forces.

The Flight Surgeon should familiarize himself with the specific parachute assemblies in use by his unit by making frequent visits to the parachute loft. Parachute riggers generally are well aware of their responsibilities, well versed in the capabilities and limitations of survival/safety equipment, and they are usually willing to discuss equipment items with interested personnel and to demonstrate their usage. The parachute loft maintains a file of ACSEBs also.

BAILOUT

The ability to exit an aircraft manually becomes problematic when indicated airspeed is in excess of 160 knots. Below this speed, the average airman is physically capable of overcoming windblast to go over the side.

The most important factor concerning successful bailout is strict adherence to prescribed procedures for egress and parachute actuation. Coupled with this, the decision to jump must be made at a safe altitude. Exact procedures for conventional bailout differ from aircraft to aircraft. The NATOPS manual for each aircraft specifies the particular exits to be used by each crewmember, the order in which crewmembers are to exit, specific tasks each man is responsible for accomplishing prior to leaving, and the egress techniques to be employed. Therefore, the NATOPS manual should be used in preparing bailout lectures or in assessing the correctness of training given by other personnel.

Some principles of conventional bailout are common to all aircraft. In all cases the escapee should visually determine that he is clear of the aircraft prior to actuating the parachute. If the escape has been initiated at medium altitudes, from 2,000 to 15,000 feet, it is desirable to delay ripcord actuation from 5 to 10 seconds. This can be estimated by counting "one thousand one, one thousand two," etc. This technique will afford a loss in momentum when the bailout has been made at high airspeed, permitting a slowdown to terminal velocity. Opening shock will be reduced as a function of reduced airspeed and lower altitude at the time of canopy opening.

Figure 14–1 shows the relationship of opening shock versus air-speed for a 28-foot canopy. Figure 14–2 shows the rate of free fall of a human body equipped with an unopened parachute at low altitudes. From this figure it can be seen that successful escape below 400 feet is unlikely since the time for deployment and canopy inflation may well take two to five seconds.

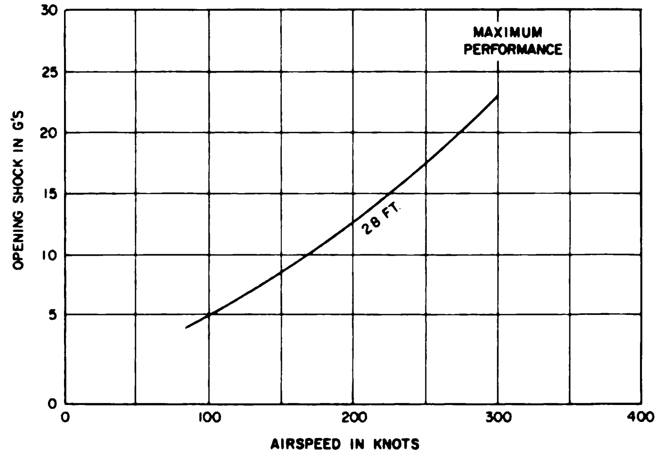


FIGURE 14–1. *Canopy opening shock versus airspeed for 28-foot-diameter parachute.*

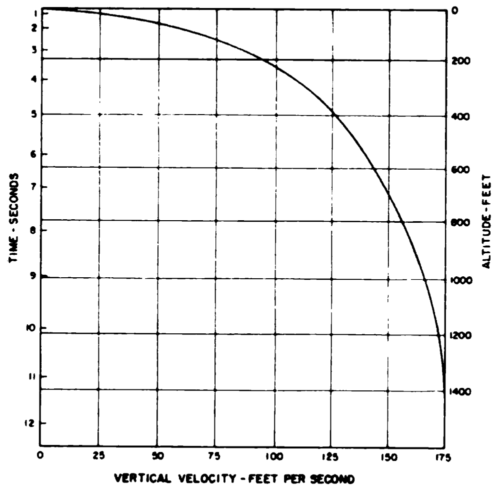


FIGURE 14–2. *Time, altitude, velocity relationships for free fall of a human body equipped with an unopened parachute.*

Problems

High-Altitude Bailout.—High-altitude bailout introduces additional factors which are detrimental to the jumper. Terminal velocity increases at altitude, with the result that parachute opening shock is greatly increased to a point where damage to the parachute structure and/or injury to the airman may result. The higher altitudes also expose the individual to low partial pressure of oxygen (hypoxia) and low temperatures.

Opening Shock.—The problem of high opening shock is the most important factor. As altitude increases, air density decreases, and terminal velocity itself increases. Terminal velocity is dependent upon the ratio of aerodynamic drag to the weight of the falling body. Aerodynamic drag is a function of air density. Therefore, at higher altitudes, the falling body falls at a faster rate to create an air drag equal to the weight of the body. Two other factors induce increased opening forces at higher altitudes. During parachute deployment, the drag created by the "streaming" chute is less at high altitude, thus less deceleration forces are applied. In addition, the increased rate of air flow and the reduced resistance to opening caused by low air density cause a more rapid deployment and inflation of the canopy. The overall effect is significant, and aircrew personnel should be familiar with the consequences of high-altitude parachute actuation. Figure 14–3 shows the rela-

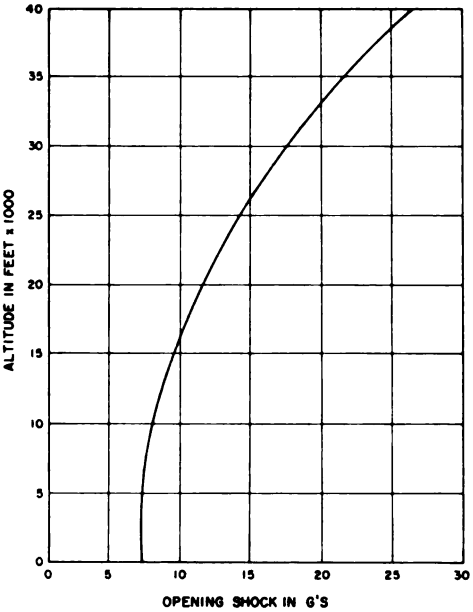


FIGURE 14-3. *Parachute opening shock in relation to deployment altitude at terminal velocity of man (28-foot canopy).*

tionship of altitude and parachute opening shock at the terminal velocity of an average crewman.

Hypoxia.—The problem of hypoxia during emergency bailout is covered in chapter 11 under "Bailout Oxygen."

Temperature.—Adequate protective clothing is required for high-altitude bailout regardless of whether the jumper free falls or makes an open parachute descent. Hypoxia will also increase susceptibility to frostbite. Gloves are especially important since finger dexterity plays an important role during parachute landing and postlanding survival activities.

Accident experience shows that problems associated with high-altitude conventional bailout are largely theoretical. This is primarily because naval aircraft which have high-altitude capability and missions are equipped with ejection seats which include automatically actuated emergency bailout oxygen supplies and barometrically actuated parachute opening systems. Thus manual over-the-side bailout occurs usually from aircraft which operate at the low and medium altitudes. Accordingly, hypoxia and low-temperature factors associated with high-altitude bailout are somewhat academic to aircrew personnel operating conventional fixed- and rotary-wing aircraft.

Parachute Descent

Technique.—To reduce the possibility of injury or entanglement during parachute deployment, certain body positions and techniques are desirable. As mentioned earlier, when time permits, while performing the "count-down," the body should be bent slightly at the waist and the feet pressed tightly together (not crossed). The chin should be positioned against the chest with the ripcord handle in view. The ripcord should be grasped with the right hand or both hands and pulled. The technique to be used with a chest pack differs slightly. The left hand should be used to hold the pack assembly while the right hand is used to pull the ripcord. As the ripcord is pulled, the head should be turned to the right side and may be further protected by placing the right forearm across the forehead and resting the face in the crook of the arm.

During free fall the recommended opening position should be maintained. Tumbling and spinning are to be anticipated. Disorientation, confusion, and nausea may accompany random tumbling and spinning. Experienced parachute jumpers are able to control body stability during planned free fall descents; however, the inexperienced jumper operating in an emergency situation is not expected to achieve a stabilized body position during descent. Accident experience indicates that many airmen, upon exposure to the effects of tumbling or spinning, actuate the parachute regardless of altitude.

The effects of nausea and disorientation, in combination with the overall psychological and physical stresses encountered during escape, frequently leave the escapee in a state of general malaise, feeling exhausted and emotionally "drained." This condition may last for a few hours after landing and points out the requirement for overtraining in the procedural aspects of post-landing survival and signalling.

Vertical Descent Velocity.—The vertical velocity at which the canopy lowers the airman is essentially a function of canopy size. The larger the canopy diameter, the lower the landing velocity, as modified, of course, by the air density changes with descent.

Table 14–1 indicates the rates of descent at sea level for various parachute canopies and for various weights. The lower weight, 160 pounds, represents the 5th percentile airman plus clothing and survival equipment. The upper weight, 280 pounds, shown for the 24- to 28-foot canopies represents the heavier individual (95th percentile) clothed in a full pressure suit and carrying support and survival equipment. Since the 26-foot canopy is not used with aircraft which require a pressure suit, the upper weight, 250 pounds, represents a large individual wearing standard flight clothing and survival equipment.

TABLE 14–1. *Rate of Descent at Sea Level for Various Size Parachute Canopies and Man/Equipment Weights*

Man/Equipment Weight	Canopy Size (Diameter) in Feet and Designation		
	24′ Martin-Baker—Rate of Descent	26′ NB-6—Rate of Descent	28′ NB-7, 8, 9—Rate of Descent
280 lbs	25 ft/sec		22 ft/sec
250 lbs		22 ft/sec	
160 lbs	20 ft/sec	18.5 ft/sec	17.5 ft/sec

The rates of descent can be equated practically to the equivalent of jumping from heights of 3 to 10½ feet onto solid ground. Parachute opening accelerations also vary as a function of canopy diameter, however, not in the way which would normally be assumed. The smaller canopies impart a greater G loading during opening than do larger ones. The larger canopy, upon deployment, streams behind the aviator, creates a loss of momentum, and takes a longer period of time to inflate than does a smaller canopy. The smaller canopy reduces momentum to a lesser degree and inflates rapidly. The difference in momentum reduction and filling time accounts for the greater acceleration imparted by the smaller canopy.

Parachutes are extremely reliable; however, the aviator may find that upon canopy deployment, the risers and suspension lines are twisted or that one or more suspension lines are across the top of the canopy. The untwisting of risers and suspension lines can be accelerated by spreading the risers apart. The "line-over" condition can normally be corrected by pulling on the suspension lines closest to the canopy skirt. The lines should slip off the canopy when pulled. Accident experience shows that pilots are often leery of attempting to correct a "line-over" and instead accept the condition and the increased rate of descent which it causes.

Oscillation during descent can be discomforting and can be dangerous during landing. Corrective action is to pull down one set of risers, which causes the canopy to slip and decreases the swinging motion. The risers are then slowly released to the normal position. This procedure increases the rate of descent and should not be attempted below 200 feet.

Parachute Landing

The airman performing an emergency parachute landing does not have the luxury of selecting his landing site, weather conditions, and the like. He may be landing in mountainous terrain where high rates of descent and harder landings will be experienced along with updrafts and downdrafts. He may be landing at night which precludes his even seeing landing hazards. In addition, he may be landing with high surface winds which impart a horizontal velocity during landing and may cause him to be dragged across the ground or water.

The proper prelanding position is mandatory if injury is to be minimized. Where altitude permits, the prelanding position should be assumed at an altitude of 1,000 feet above ground level. Both arms should be outstretched above the head with the hands firmly grasping the risers. The knees should be slightly bent and the feet held together (not crossed). The jumper should direct his view at a 45-degree angle to the ground. This line of vision will preclude the anticipation of surface contact and the retraction of legs which becomes almost an involuntary act if the jumper looks straight down.[1] Statistics concerning night landings indicate that the chances of injury are less than during daylight hours which is ascribed to the fact that the landing impact can not be anticipated and less anticipatory tensing and leg retraction occurs.

The landing should be made on the balls of both feet and the body should be permitted to collapse in the direction of parachute movement.

The parachute must be collapsed to preclude dragging. This is done by pulling the lower risers which causes the canopy to "spill." Some parachute harnesses are equipped with a mechanical quick-release mechanism which permits the release of the canopy from the harness assembly.

When the landing is to be made in trees, the arms should be folded in front of the face and the legs held together (not crossed). This position should be maintained until all vertical movement ceases.

Water landings present additional hazards. It is extremely difficult to judge height above water. Therefore, no attempt should be made to release from the harness just prior to landing. Drowning from dragging is a major problem and can happen to the best of swimmers even with relatively light surface winds. Accordingly, the airman must release canopy releases immediately upon entering the water or shed the entire harness assembly. Training for this situation and overall conventional bailout is discussed in a later section.

Bailout Statistics

Table 14–2 shows the cumulative injuries resulting from bailouts by aircraft model for a recent ten-year period. The aircraft models shown parenthetically were not in service during the final period covered by this table.

Table 14–3 depicts cumulative injuries resulting from bailouts according to altitude.

Table 14–4 relates the same bailout occurrences and injuries to aircraft speed at time of abandonment. All three tables are for the same ten-year period.

EJECTION

The advent of jet aircraft made the ejection seat a necessity for successful escape. Severe windblast prevented individuals from clearing the aircraft, caused premature deployment of parachutes, and caused primary injury. Violent oscillations and spinning of aircraft imparted acceleration forces which prevented escape. Extremely high sink rates in the power-off configuration prevented low-altitude escape. To rectify this situation, ejection seats were installed in most Navy jet aircraft, although some are equipped with an escape chute or slide which provides an exit for bailout which is protected from windblast.

Ejection Seat

A typical ejection seat consists of a sheet metal seat bucket, back, and headrest. Attached to the back of the seat are guide rollers which ride on a guide rail assembly attached to primary aircraft structure. The guide rails provide directional stability to the ejection seat during its initial movement. A catapult tube attached to the seat back fits over another tube attached to the ejection gun and cockpit structure. The ejection gun contains an explosive charge or series of charges which, when fired, catapults the upper tube and the attached seat up the guide rails and clear of the aircraft.

TABLE 14–2. *Cumulative Injuries Resulting from Bailouts According to Model, 1953–1963*

Model Aircraft	Total	Resulting Injury to Personnel									
		Fatal		Critical		Severe/ Serious		Moderate/ Minor		None	
		No.	%	No.	%	No.	%	No.	%	No.	%
A–1	112	17	15	1	1	17	15	48	43	29	26
(AF)	3					1	33	2	67		
(AU)	1							1	100		
A–2	10					1	10	6	60	3	30
A–3	69	5	7			6	9	50	72	8	12
(FG)	3			1	33	1	33	1	34		
F–1	1							1	100		
F–8	1					1	100				
F–2	1							1	100		
F–3	2	2	100								
F–10	24	3	13			1	4	12	50	8	33
F–6A	1							1	100		
(F4U)	13	3	23			5	39	3	23	2	15
(F6F)	11	1	9			1	9	7	64	2	18
(F8F)	7	2	28			2	29	3	43		
F–9	2							2	100		
B–26J	10	1	10					4	40	5	50
P–2	23					2	9	8	35	13	56
P–5	12	1	8			1	8			10	84
C–119	14	6	43					6	43	2	14
S–2	3	2	67			1	33				
TC–45J	8							3	38	5	62
(SNJ)	62	6	10	1	2	6	10	28	45	21	33
(TBM)	7					1	14	4	57	2	29
T–28	33	2	6			4	12	17	52	10	30
T–33B	4	1	25					3	75		
T–34B	3							1	33	2	67
UH–25	2	1	50			1	50				
UH–2A	1							1	100		
Totals	443	53	12	3	1	52	12	213	48	122	27

From *Emergency Airborne Escape Summary—CY 1963*, Naval Aviation Safety Center, Aeromedical Department.

TABLE 14–3. *Cumulative Injuries Resulting from Bailouts According to Altitude, 1953–1963*

Altitude	Total	Resulting Injury to Personnel									
		Fatal		Critical		Severe/ Serious		Moderate/ Minor		None	
		No.	%	No.	%	No.	%	No.	%	No.	%
0–999	51	20	39	......	......	7	14	17	33	7	14
1000–1999	50	7	14	1	2	2	4	19	38	21	42
2000–2999	82	6	7	......	......	11	14	43	52	22	27
3000–4999	105	3	3	1	1	8	8	58	55	35	33
5000–9999	111	7	6	1	1	17	15	56	51	30	27
10,000–19,999	27	3	11	......	......	2	7	17	63	5	19
20,000–29,999	5	......	......	......	......	1	20	3	60	1	20
Unknown	12	7	58	......	......	4	33	......	......	1	9
Totals	443	53	12	3	1	52	12	213	48	122	27

From *Emergency Airborne Escape Summary—CY 1963*, Naval Aviation Safety Center, Aeromedical Department.

TABLE 14–4. *Cumulative Injuries Resulting from Bailouts According to Speed, 1953–1963*

Speed	Total	Resulting Injury to Personnel									
		Fatal		Critical		Severe/ Serious		Moderate/ Minor		None	
		No.	%	No.	%	No.	%	No.	%	No.	%
0–99	28	1	4	......	......	1	4	17	63	9	32
100–149	173	15	9	1	1	22	13	75	43	60	34
150–199	99	7	7	1	1	8	8	54	55	29	29
200–249	40	4	10	......	......	5	12	24	60	7	18
250–299	20	1	5	......	......	4	20	12	60	3	15
300–349	9	3	33	......	......	3	34	2	22	1	11
350–399	2	......	......	......	......	......	......	2	100	......	......
400–449	2	1	50	......	......	......	......	1	50	......	......
450–499	......	......	......	......	......	......	......	......	......	......	......
500–549	2	......	......	......	......	......	......	2	100	......	......
Unknown	68	21	31	1	2	9	13	24	35	13	19
Totals	443	53	12	3	1	52	12	213	48	122	27

From *Emergency Airborne Escape Summary—CY 1963*, Naval Aviation Safety Center, Aeromedical Department.

There are many variations in specific ejection seat designs and capabilities. There are essentially three ejection seat types now in use: Standard, Martin-Baker, and rocket.

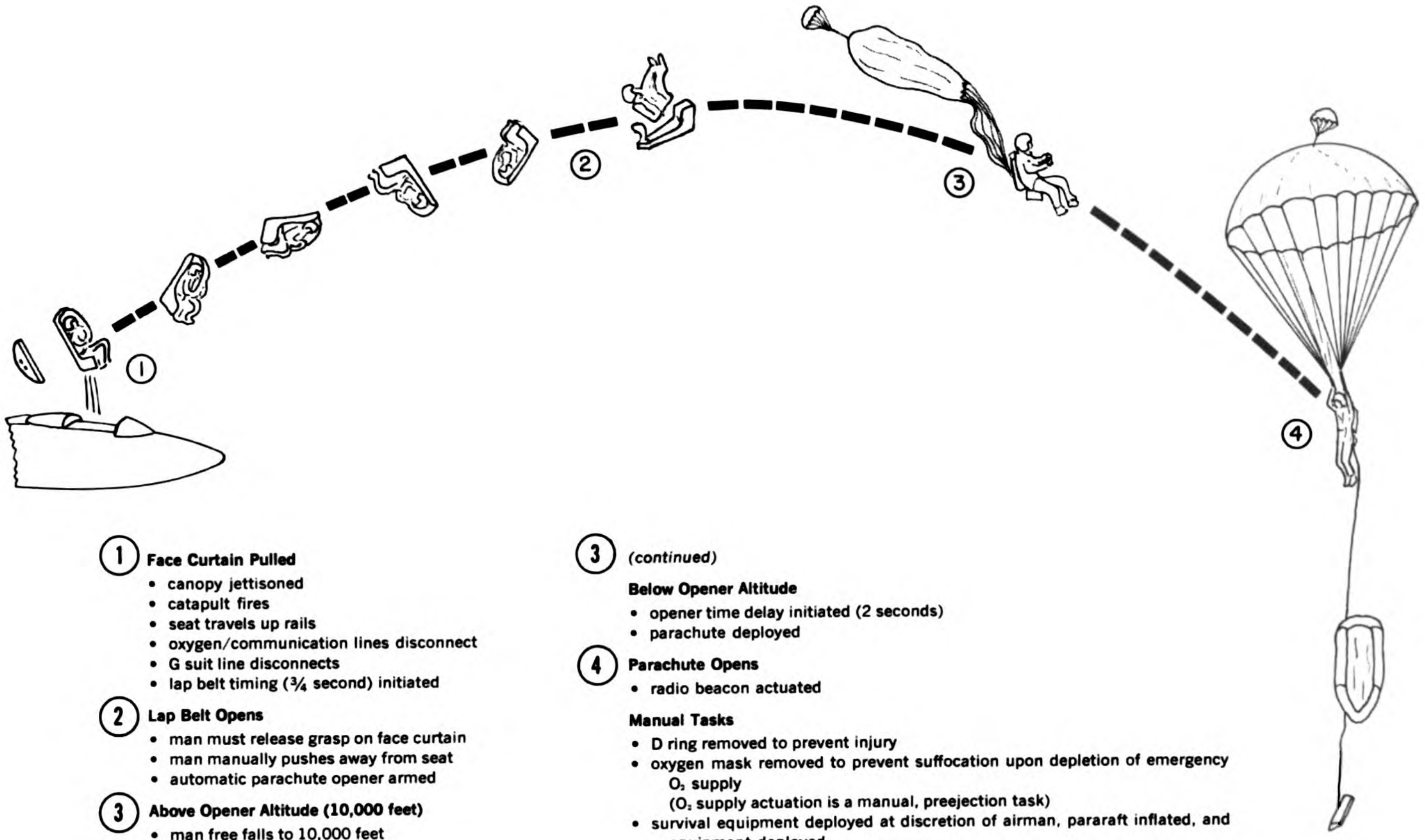
The Standard Seat.—At one time all Navy aircraft had a single type of ejection seat and utilized a single catapult (the NAMC type) and a single cartridge type. Taken together, these three items constituted the "standard" ejection seat system.

The Martin-Baker system represented a marked improvement over the standard system for low-altitude ejection. It was purchased and retrofitted into most naval aircraft. Those aircraft in which a retrofit was not performed for one reason or another retained the standard seat, and the zero-delay lanyard was installed to improve low-altitude performance. It will be discussed later.

The T–33B, a U.S. Air Force aircraft, still has the armrest and trigger method of firing the seat. The raising of left armrest arms the seat and locks the entire reel. Raising of the right armrest jettisons the canopy and exposes the attached seat trigger which is then squeezed to fire the seat. The T–33 is obsolescent and has many other systems unique in naval aircraft, especially its oxygen supply system and fuel tank arrangement.

Figure 14–4 graphically depicts the sequence of events which occurs during the use of the conventional ejection seat system. The aviator positions himself in the seat, buttocks well back, head against headrest, body erect. The feet are placed firmly in footrests or "stirrups." This position minimizes stress on the anterior portions of the vertebrae during seat firing and reduces the flailing of the lower limbs upon entry into the windblast. The face curtain located at the top of the headrest is grasped with the hands, palms toward the head. The curtain is pulled down over the face with elbows held in. Initial movement of the curtain fires a canopy release mechanism, the final movement fires the seat catapult. The face curtain firing technique serves three purposes: the action aids in achieving correct body positioning, prevents upper extremity flailing, and protects the head and face from windblast during canopy release and entry into the airstream. It is recognized that under asymmetric flight conditions, when G forces exceed 6 to 8 G (even when wearing a G suit), and in cases where the occupant has been injured, the face curtain presents difficulties. Accordingly, a secondary firing actuator handle is located between the legs at the forward edge of the seat bucket.

As the seat starts up the rails, the automatic lap belt system is armed (timing commences), and oxygen/communication disconnects are separated.



(1) **Face Curtain Pulled**
- canopy jettisoned
- catapult fires
- seat travels up rails
- oxygen/communication lines disconnect
- G suit line disconnects
- lap belt timing (¾ second) initiated

(2) **Lap Belt Opens**
- man must release grasp on face curtain
- man manually pushes away from seat
- automatic parachute opener armed

(3) **Above Opener Altitude (10,000 feet)**
- man free falls to 10,000 feet

(3) *(continued)*

Below Opener Altitude
- opener time delay initiated (2 seconds)
- parachute deployed

(4) **Parachute Opens**
- radio beacon actuated

Manual Tasks
- D ring removed to prevent injury
- oxygen mask removed to prevent suffocation upon depletion of emergency O_2 supply
  (O_2 supply actuation is a manual, preejection task)
- survival equipment deployed at discretion of airman, pararaft inflated, and equipment deployed
- landing position assumed

FIGURE 14–4. *Ejection sequence of conventional ejection seat system.*

Leaving the guide rails, the seat pitches forward and commences tumbling in head-over-heels fashion. The amount and rate of tumbling are largely dependent on the ejection airspeed.

After ¾ second, the lap belt automatically separates, leaving the occupant free to push the seat away. Depending on the flight attitude of the seat at this time and the ejection airspeed, the seat will either separate due to differential drag or will be kicked away by the occupant. Seat separation arms the automatic parachute opener.

Above an ejection altitude of 10,000 feet, the barostat in the automatic parachute opener prevents automatic parachute opening. Below 10,000 feet, the automatic opener actuates after a two-second delay. The delay is required to permit a decay of momentum when the ejection is made at high speed and below 10,000 feet. Otherwise, injury or damage could occur to the escapee or the parachute.

Barostatic parachute opening devices are universally installed in ejection seat-equipped jet aircraft but have some limitations. The worst is that if the terrain is at a higher altitude than the setting of the barostat, the aviator will not get automatic parachute deployment. In one case, an aviator ejected at 14,000 feet, normally considered a safe altitude for ejection. However, he happened to be over a mountain range with terrain at 12,000 feet. The barostat was set to deploy the parachute at 10,000 feet. He did not, of course, get parachute deployment before hitting the ground. The aviator flying over mountainous terrain must keep this in mind and deploy his parachute manually as soon as possible after ejecting. Another way of attacking this problem is to reset the parachute barostat to open at a higher altitude, but this is a matter of squadron doctrine, and must be in accordance with the NATOPS manual.

After parachute deployment and inflation, the jumper removes his oxygen mask if below 10,000 feet to prevent breathing difficulty upon exhaustion of the emergency oxygen supply. In some systems, the force of the parachute opening is used to pull a lanyard connected to a survival radio. During descent a signal is emitted on the ultrahigh frequency (UHF) designated as emergency (243 mc.) to alert rescue forces and to guide them to his location. The zero-delay lanyard is utilized in standard seat installations. It bypasses the time-delay feature of the automatic parachute opener while operating at low altitudes. A lanyard attached to cockpit structure is manually attached to the parachute "D" ring prior to takeoff or when entering the landing pattern. Upon ejection, parachute deployment is immediate.

The zero-delay lanyard has the disadvantage of being manual. If the pilot forgets to unfasten it after takeoff and ejects at high speed or altitude, the results could be catastrophic due to the resulting G forces and parachute damage. Conversely, if he fails to refasten it, he has no low-altitude capability upon reentering the landing pattern.

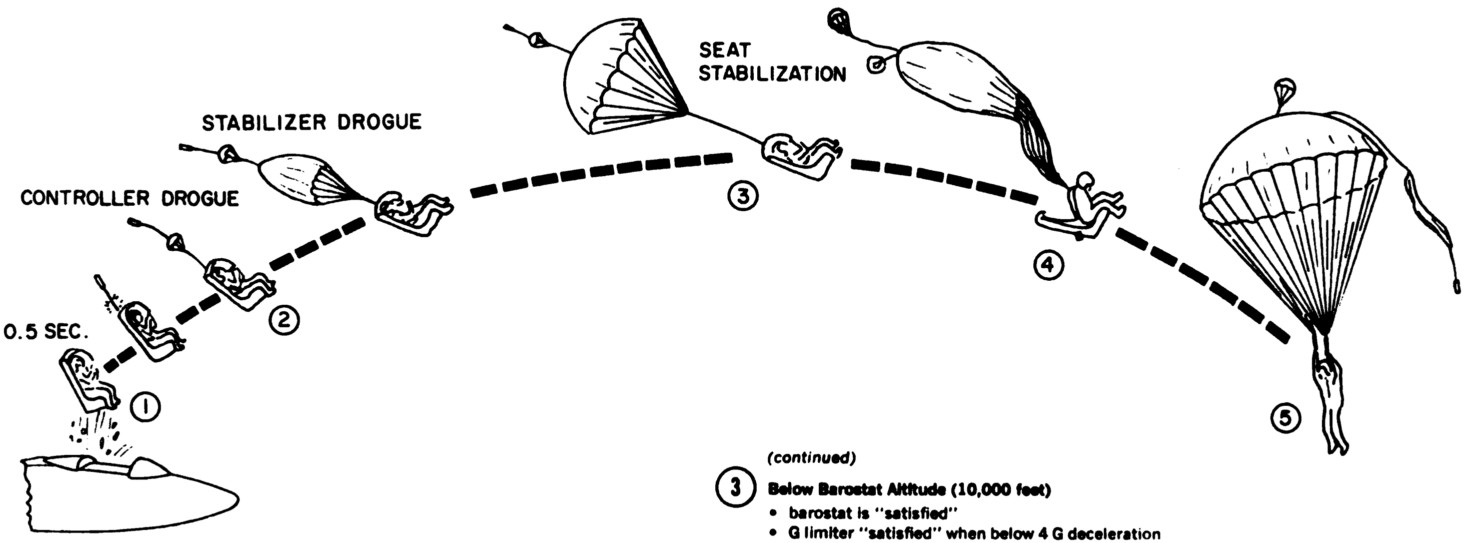
The Martin-Baker Ejection Seat.—The Martin-Baker ejection seat system is named for the British firm which manufactures the seat and is responsible for many pioneering developments in the emergency escape field. The key feature of a Martin-Baker system is the use of a multiple (three) cartridge that is fired in series and produces a higher ejection velocity but does not increase peak G or rate of onset as a larger single cartridge would. A mechanical device called a "G limiter" senses deceleration forces on the man-seat combination and prevents parachute deployment until the decelerative force field is within tolerance of man and equipment (parachute). Figure 14–5 shows the sequence of events for a typical Martin-Baker system.

Upon making the decision to eject the aviator positions himself in the same manner as described under "conventional ejection seat." Unlike the conventional system, automatic leg retraction and restraint are provided. Upon boarding the aircraft, leg garters are attached to a cable takeup system located in the seat bucket. The system provides freedom of the lower limbs during normal flight activities. Upon actuation of the primary (face curtain control) or secondary (ejection control) the cables attached to the leg garters are forcibly retracted by a power reel system and restrained in proper position until the seat separation mechanism is triggered.

In many Martin-Baker systems the aircraft canopy is deliberately not removed prior to seat firing. Rather, the headrest of the seat is provided with an impact device which shatters the canopy as it contacts the canopy structure. This eliminates the time delay for canopy removal system malfunctions.

Immediately after the ejection catapult is fired, a drogue gun located on the headrest fires and a metal slug propels a 22-inch controller drogue parachute into the airstream. In turn, a 60-inch stabilizer drogue is extracted by the controller drogue. The drogues aid in stabilizing the seat and are connected to the G limiter which, in turn, is attached to the seat proper.

When the decelerative field is less than 4 G, the G limiter is satisfied and the main parachute is deployed, providing the altitude is below the limit set for the barostat (10,000 feet). Thus, two factors must be satisfied for the main parachute deployment: the G limiter and the altitude sensor. At high



① **Face Curtain (or secondary ejection handle) Pulled**
- catapult charge fires
- seat travels up rails
- oxygen/communication lines disconnect
- G suit connection disconnects
- emergency oxygen supply activated
- leg retraction initiated

② **Drogue Gun Fires**
- slug pulls out 22-inch chute
- 22-inch chute extracts 5-foot drogue chute

③ **Above Barostat Altitude (10,000 feet)**
- free fall in seat with drogue deployed
- G limiter "satisfied" when under 4 G deceleration (300 knots)
- barostat "satisfied" when below setting (10,000 feet)
- time delay (1 ¾ seconds) initiated

(continued)

③ **Below Barostat Altitude (10,000 feet)**
- barostat is "satisfied"
- G limiter "satisfied" when below 4 G deceleration
- time delay (1 ¾ seconds) initiated

④ **Time Delay Completed**
- personnel recovery parachute deployed
- face curtain released
- leg restraint released
- man/equipment restraint released
- man pulled out of seat

⑤ **Personnel Recovery Chute Opens**
- radio beacon actuated

Manual Tasks
- D ring removed from pocket to prevent injury
- oxygen masks removed to prevent suffocation when emergency O_2 is depleted
- survival equipment container actuated at airman's discretion during descent
- pararaft deployed and inflated
- landing position assumed

FIGURE 14-5. *Martin-Baker ejection sequence.*

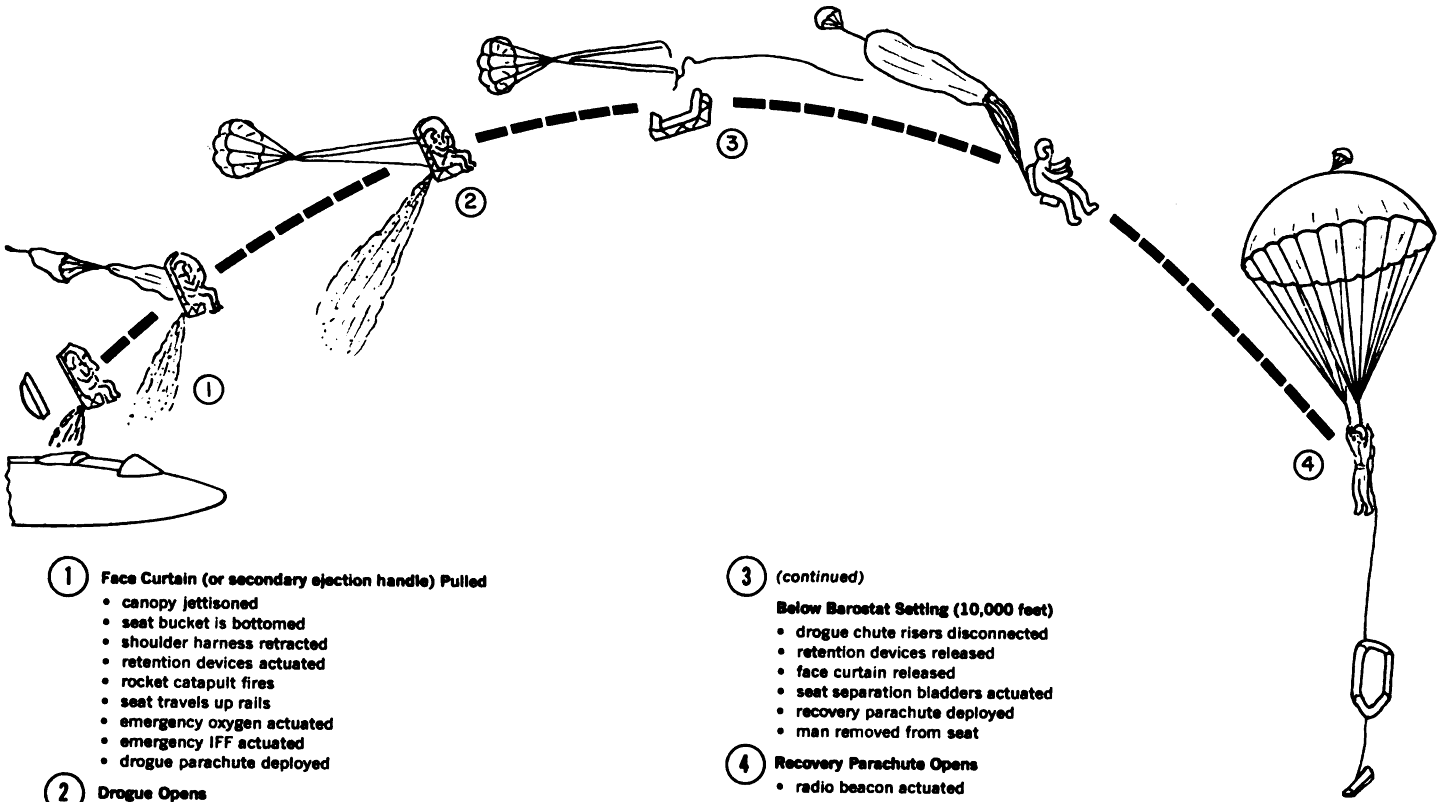
altitude, above 10,000 feet, after the G limiter has been satisfied, the barostat will prevent parachute actuation and the aviator descends in the seat (drogue chute deployed). Upon reaching 10,000 feet, the barostat is satisfied and the recovery parachute sequence is commenced.

At low altitude (below 10,000 feet) the barostat is satisfied immediately; however, the G limiter will prevent deployment until the 4-G "gate" is sensed by the G limiter.

After both G and altitude gates are satisfied, a time delay system provides additional protection. The delay is 1¾ seconds.

The deployment of the main recovery parachute is accompanied by additional system functions. The seat separation system is actuated, the body restraint harness is separated from the seat structure, leg restraint cables are released, and face curtain is released. The deployment of the main parachute then provides the separation force differential between man and seat and precludes the possibility of man-seat collision or entanglement of the seat in the parachute assembly, which sometimes occurs with conventional seat systems. The man then descends under the recovery parachute with survival equipment attached. Activities such as communication and oxygen disconnect and automatic emergency oxygen supply actuation occur during the initial upward movement of the seat, and the survival radio beacon is actuated upon parachute deployment.

Rocket-Augmented Ejection Seat.—Some ejection seats utilize rocket propulsion in addition to the cartridge-type ejection gun. A rocket attached to the seat structure is ignited as the seat is being propelled upward by the cartridge gases. The rocket assist provides a number of desirable features. The rocket propulsion acts as a sustainer, maintaining propulsion after the normal catapult tubes have separated. A higher ejection trajectory results. In addition, the thrust of the rocket is aligned with the center of gravity of th man-seat combination, which affords stability during the initial flight of the seat. The rocket thrusts upward and forward so that additional velocity is attained, which causes the parachute to inflate more rapidly at low ejection airspeeds and in a more predictable manner. Figure 14-6 shows rocket seat sequencing. Rocket acceleration is less abrupt than that with multiple cartridges; thus, peak G is reduced for an equivalent ejection height. However, the reduction in G, by increasing time, exposes the occupant to deceleration forces for a greater period of time. Cartridge-type ejection typically occurs in a time period of 0.2 second and rocket-assist ejection, 0.5 second. During rocket ejection windblast overpressure and erosion effect are more severe and



① **Face Curtain (or secondary ejection handle) Pulled**
- canopy jettisoned
- seat bucket is bottomed
- shoulder harness retracted
- retention devices actuated
- rocket catapult fires
- seat travels up rails
- emergency oxygen actuated
- emergency IFF actuated
- drogue parachute deployed

② **Drogue Opens**
- seat "flies" up and forward, stabilized by rocket thrust vector through man/seat center of gravity and drogue chute

③ **Above Barostat Setting (10,000 feet)**
- man descends in drogue-stablized seat
- emergency O_2 supplied

③ *(continued)*

Below Barostat Setting (10,000 feet)
- drogue chute risers disconnected
- retention devices released
- face curtain released
- seat separation bladders actuated
- recovery parachute deployed
- man removed from seat

④ **Recovery Parachute Opens**
- radio beacon actuated

Manual Tasks
- D ring removed from harness
- oxygen mask removed
- survival kit deployed at discretion of airman pararaft inflated, equipment deployed
- landing position assumed

FIGURE 14–6. *Rocket catapult ejection sequence.*

separation of the ejectee from the seat takes longer. The increased low-level, low-airspeed capability with the rocket-assist seat, however, is considered to be more important than the increased windblast disadvantages. The rocket system also permits more effective use of stabilizing fins and parachutes during initial entry into the airstream due to its predictable, continuous thrust vector.

All other features of rocket-augmented systems are essentially the same as discussed under the other seat systems.

The rocket system solves the problem imposed by human tolerance to impact acceleration and its restriction on escape trajectory height. Thus, high trajectories can be achieved within the limits of human tolerance to impact accleration. Some rocket seats are capable of saving the occupant under "zero-zero" conditions (zero velocity and zero altitude).

Capsular Escape Systems

For some time, escape system designers have believed that a closed escape system is the only means of successfully rescuing an occupant from a vehicle traveling at supersonic speeds.

The closed system obviates the problems associated with windblast pressure, erosion effects on equipment and clothing, and temperature, and would permit the discarding of many of the items of equipment which the flyer feels affect his ability to perform his primary tasks of operating the aircraft.

Closed escape systems are basically of two types: one, an enclosed ejection seat sometimes referred to as a homoform system, and two, a capsule consisting of the aircraft structure itself. The latter type may be a jettisonable nose section or any portion of the vehicle which is occupied. The engineering problems relative to both types are formidable. Research and development effort has been ongoing for both types since the late 1940s. The homoform system requires a means of rapidly enclosing the seat and occupant and must not restrict normal flight activities when in the stowed or open configuration. Early designs were rather bulky and the possibility of retrofitting such a device into existing aircraft structure appeared remote. Stabilization, recovery, and weight problems also were experienced.

Such a system finally was developed and approved for installation in the Air Force B–58 aircraft and other systems in the XB–70.

Early capsules of the cockpit-section type were plagued by weight, ineffective separation techniques, and stabilization recovery problems. In general, it appeared that the occupied cockpit area would first have to be designed as an escape capsule and the remaining portion of the aircraft would have to conform to the capsule system. This imposed severe constraints on primary aircraft design and operation capabilities and was considered untenable.

A relatively recent technological development, the flexible linear-shaped charge, has made the capsular concept practical. This invention consists of a fast-burning chemical powder encased in a metal sheath or tubing. The tubing is extremely malleable and can be readily formed into eccentric shapes. The tubing is placed in position, circling or outlining the portion of primary aircraft which is to be used as a capsule. Upon actuation, ignition occurs and the powder within the tubing burns at a fast rate (25,000 feet per second), cutting through the material adjoining the tubing. This provides a method of instantaneously "chopping out" a capsule from almost any portion of the aircraft. The resulting capsule is then "boosted" free of the remaining aircraft structure with a rocket propulsion system and recovered by parachute. This concept is commonly referred to as "chop-boost-recover." The F-111 is the first operational aircraft to be equipped with such a system.

A capsule system offers the advantages of omnienvironmental protection and minimization of the tumbling and spinning associated with free fall descent. Landing stresses are minimized and predictable (parachute landing technique is not a factor). The capsule contains all necessary supplemental survival and signalling equipment, provides a shelter for land survival, and is buoyant. One of the major advantages is the lack of encumbrance which the capsule provides to the aviator, freeing him of the multitude of protective devices and clothing evident in contemporary open escape systems.

Dynamics of Ejection

Peak Acceleration and Rate of Onset.—If the engineer were to ignore human tolerance to acceleration, it would be a simple matter to calculate the total ejected mass and provide an explosive charge capable of achieving the necessary velocity within the stroke of the catapult and meeting the trajectory requirements. The design criteria for ejection seats are: a maximum of 20 G upward acceleration with a rate of onset of not more than 250 G per second. Present systems are well within these limits. Table 14-5 shows the relationship of peak G and rate of onset for different ejection seat systems. The lower values for peak G and rate of onset represent the acceleration for a 95th percentile man; the upper figure a 5th percentile man. For the rocket catapult, the upper peak-G values are for the cartridge or boost phase, and the lower values are for the rocket or sustainer phase of the ejection sequence.

TABLE 14–5. *Peak Acceleration and Rate of Onset Values for Various Ejection Seat Systems*

Ejection System	Peak G	Rate of Onset G's per second
Conventional NAMC	17–20	200–250
Martin-Baker (3 cartridge)	15–18	250
Rocket Catapult	12–13 8–9	140–180

It should be understood that many factors influence the actual values which a catapult will give for a specific ejection. Propulsion devices are affected by temperature, the total weight of the man-seat assembly (which varies with differences in personnel and equipment and clothing worn), the airspeed at time of ejection, the altitude (air density) of ejection and aircraft attitude at firing, and the tumbling of the assembly in the airstream, will all cause variation in ejection forces actually encountered.

Stapp (1958) reports exposures of human subjects to 30 and 33 G at a rate of onset of 500 G per second. However, these encounters were under ideally controlled conditions.

From a practical view, the G forces encountered by escapees may exceed the stated design limits. This is due to the complex mechanical behavior of various parts of the body in relation to each other and the relation of the body to the seat when the man-seat system is subjected to ejection forces. The man-seat system is a complex mechanical system of rigid and semirigid masses connected by elastic elements. When the ballistic force is applied, internal interactions cause time lags as elastic elements absorb energy and then release the absorbed energy as the compression force ceases. This release causes additional peaks of acceleration sometimes called "dynamic overshoots."

Early ejection seat experience indicates the importance of this factor. Subjects using thick elastic seat cushions suffered injuries more severe than would be expected from the accelerometer data taken from the seat proper. Instrumentation of anthropometric dummies, which, incidently, do *not* have the same internal dynamics as the human body, revealed that the cushioning caused extreme levels of dynamic overshoot. Upon ejection the resilient seat cushion readily compresses, the seat is well on its way before the man has started upward, yet the final velocity of man-seat assembly is the same. The man then has been accelerated to peak velocity in a shorter time period than the seat assembly, resulting in a greater rate of G onset in the man.

This points out the importance of cautioning flight crews not to improvise equipment which might appear to provide more comfort (or improve sitting position) since the unauthorized and seemingly unimportant item might well cause serious injury in event of ejection.

Dynamic overshoot occurs also within the body. If the time-force characteristics of acceleration and the decay rate are in harmonic resonance with the natural frequency of the man-seat system, severe overshoots can be produced within the body. Latham (1957), in a study of body ballistics using various types of seat cushions, found that accelerations of less than 0.2 second and 400 G per second rate of change can produce a maximum acceleration overshoot in the body. A slight change in the acceleration period, even 0.03 or 0.04 second, will result in a minimum overshoot condition. This was considered when multiple charges and rocket augmentation were initially contemplated for escape systems.

Back Injuries During Ejection.—Vertebral fractures occurring during ejection have become a frequent major injury in aircraft accidents. Table 14–6 shows the incidence of vertebral fractures as currently experienced in Navy jet aircraft accidents. A variety of causal factors are ascribed to these injuries including position of the body at time of ejection; varied tension of restraint harness; inverted flight conditions at time of ejection; the technique used in pulling the face curtain (pulling out rather than down, causing forward arching of the back); improper seat and seat-back cushioning; discrepancy between body center of gravity and the upward and backward (approximately 18° from vertical) thrust line of the catapult; and through-the-canopy ejection.

Other Stresses of Ejection.—The accelerations imposed on the spinal column during ejections represent only one of several stresses to which the ejectee is exposed in very short time periods. Within milliseconds of receiving the ejection gun acceleration, the individual enters the windstream, not as a sudden total exposure but as a relatively gradual partial exposure. As the ejection seat emerges from the cockpit there is a marked differential pressure exerted on the part of the body exposed to windblast as compared with the

TABLE 14–6. *Distribution of Individuals Suffering Vertebral Fracture by Type Accident, and Year. Jets, U.S. Navy**

Fiscal Year	Ejections			Collision Water			Collision Ground			Bailout			Total		
	#	At Risk	Rate/ 100	#	At Risk	Rate/ 100	#	At Risk	Rate/ 100	#	At Risk	Rate/ 100	#	At Risk	Rate/ 100
JETS:															
1959	9	134	6.72	1	75	1.33	8	386	2.07	0	18	0	18	613	2.93
1960	11	118	9.32	3	64	4.68	8	310	2.58	0	17	0	22	509	4.32
1961**	12	118	10.17	0	65	......	5	326	1.53	0	12	0	17	521	3.26
1962	14	136	10.29	1	40	2.50	6	277	2.17	0	9	0	21	462	4.54
1963	16	110	14.54	0	41	......	9	273	3.30	1	14	7.14	26	438	5.94
Total	62	616	10.06	5	285	1.75	36	1,572	2.29	1	70	1.42	104	2,543	4.09

*Excluding injuries multiple extreme, but including injuries from *all causes* during accident, as determined by Flight Surgeon reporting the accident.

**Excludes two fatal cases of ejection vertebral fx in which bodies were dragged ½ mile by parachute but which were not classified as having injuries multiple extreme.

(Ewing, 1966a.)

part still protected. While this differential ram air pressure exists only for very short periods of time, it nevertheless does exist and can be the cause of fatal injury if prolonged. In some instances, initial exposure of the helmeted head to the windblast has caused the helmet to act as a sail causing fracture of the hyoid bone as the helmet chinstrap is suddenly impacted against it. Crash helmet design must take this factor into consideration. Also all parts of the helmet, oxygen mask, oxygen mask suspension, minireg, oxygen hose, and helmet visor must be designed, constructed, and tested for ability to withstand windblast in excess of 500 mph.

In a few recent accidents, the question of helmet rotation (around the axis of the chinstrap attachment bolts) during exposure to windblast has been raised. If such rotation occurred, one would expect to find posterior compression fractures of cervical vertebrae, with or without cord injury. Such injuries have been noted in isolated instances but may have been due to other causes. The problem may or may not exist.

After the initial + Gz acceleration of the gun, and the differential ± Gx acceleration of "gradual" entry into windblast, the entire body and seat combination is subjected to the − Gx deceleration due to ram air from windblast.

This force is proportional to the surface area of the man-seat combination and to the differential velocity of the man-seat combination and the air in which it moves.

Dynamics of Postejection Phase

Once the ejection seat has entered the airstream, several additional factors are encountered. These are: windblast, deceleration, thermal effects, and tumbling (rotational stress).

Windblast.—The dynamic pressure of the airstream encountered as the ejection seat exits the cockpit is dependent on the velocity of the air and air density. Thus, both the airspeed and altitude at time of ejection are important variables. The higher the airspeed and the lower the altitude, the greater will be the air pressure applied to the man-seat combination. For all practical purposes, the pressure (stated in pounds per square foot) is the density of the air (in slugs per cubic foot) times the velocity of the air (in feet per second), squared. The important fact here is that air pressure increases by the square of the velocity. Therefore, when possible, pilots nose up their aircraft prior to ejection to reduce airspeed and increase altitude. The additional altitude is also essential to the success of low-level escape.

The abrupt entry into air at high speed causes formidable stresses. For example, at an altitude of 5,000 feet and at a true airspeed of 600 knots (.9 Mach), the ram pressure encountered is 1,240 pounds per square foot. This means that a man presenting approximately six square feet of frontal area will receive a total pressure of 7,440 pounds or approximately 3½ tons of pressure. Stomach inflation due to the face being uncovered has occurred; lung damage, however, has not been encountered. The wearing of an oxygen mask and use of the face curtain method of actuation attenuates this hazard.

The major factor concerning windblast per se (as opposed to the deceleration) centers about the effect of the air pressure on the limbs and protective clothing and equipment. The high drag characteristics of the limbs and man's inability physically to control arm and leg movement against flailing can cause injury. The use of leg and thigh garters in the Martin-Baker ejection system and other leg retention and restraint devices on other seats diminishes this factor. The grasping of the face curtain provides some relief from total arm flailing. However, there have been instances of the elbows moving outward and away from the trunk. This "butterflying" can cause both arm and shoulder injuries, and has done so.

Another factor, sometimes termed "windblast erosion" is the effect of the air pressure on protective clothing and equipment. In the past, clothing has been torn, shoes pulled from the feet, helmet visors shattered, helmets lost, and parachutes prematurely deployed, the latter usually with fatal results.

From the experience gained in wind tunnel and rocket sled tests of personal flight and survival equipment, survivor accounts, and medical officers' reports, many improvements have been made to enhance the integrity of equipment for high-speed, low-altitude ejection. This points up the importance of adequate accident reporting even in cases where the loss or malfunction of an item of equipment was not a detrimental factor.

Deceleration.—The most formidable factor encountered in high-speed escape is the decelerative force experienced as the man-seat assembly enters the airstream. For certain combinations of airspeed and altitude the deceleration force will be greater than that encountered during a crash where the aircraft collides with ground or water. The duration of the G forces may in fact be 10 to 20 times greater than that experienced during a crash situation.

When a condition of random tumbling or spinning exists, the pattern of mechanical forces sustained by the body becomes extremely complex, almost beyond contemplation, let alone computation.

Goodrich (1956) has calculated the relationship of calibrated airspeed, altitude, and Mach number on deceleration force. Results are presented in figure 14–7. The calculations are based on a stable seat system with no tumbling or spinning. The values shown are for a particular seat system and should not be considered as absolute values. For any particular system the following factors affect the rate of onset, peak G, and duration of deceleration:

V—Velocity at time of ejection
P—Air density
A—Frontal area exposed to airstream
W—Weight of the ejected mass (man and seat)
C_d—Coefficient of drag of the occupied seat.

Goodrich also indicates that G forces show an exponential increase with velocity. This reinforces the desirability of decelerating an aircraft prior to ejection when circumstances permit. For illustrative purposes, consider two escape situations identical except for airspeed. Pilot A is at an airspeed of 200 knots, pilot B at 230 knots. Pilot B potentially may be exposed to a decelerative force which is 50 percent greater than A's.

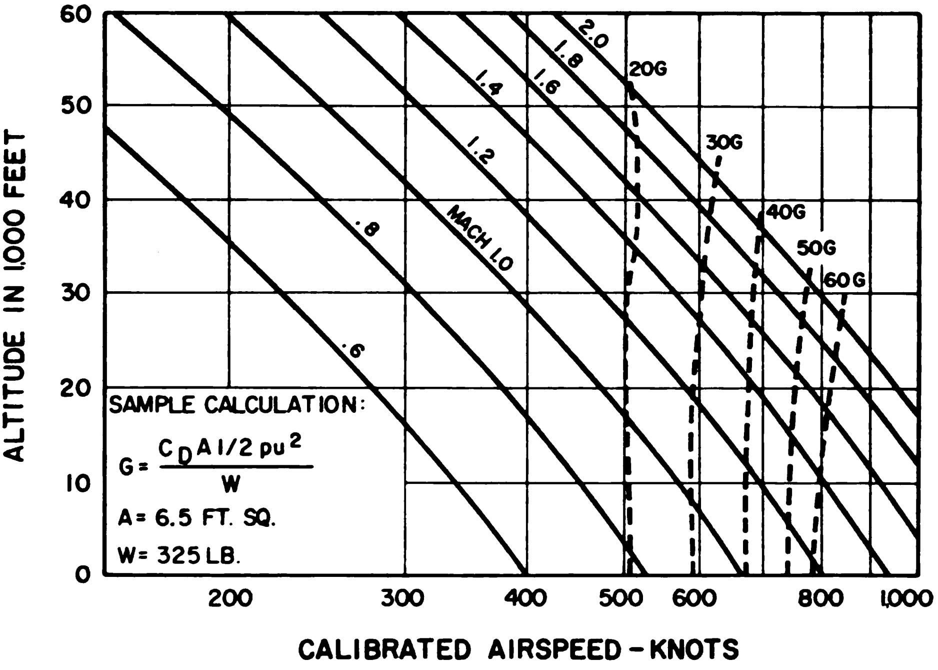


FIGURE 14-7. *Magnitude of G force as a function of altitude, calibrated airspeed, and Mach number* (GOODRICH, 1956).

The duration of the deceleration force is dependent on air density. The deceleration period increases with an increase in altitude. At 40,000 feet the deceleration period will be approximately twice that experienced at sea level air density. Figure 14-8 shows this relationship.

It is generally agreed that successful open-seat ejection becomes problematic as indicated airspeed approaches 600 knots. Injuries do occur at much lower airspeeds, and no set value for no-injury escape has been established because of the variety of factors involved, mechanical and human. Designers are constantly striving for better escape capability, more in concert with the requirement to deliver an escapee to the ground in a physical condition compatible with postlanding activities: survival, escape, and evasion.

High Temperature Exposure.—Accident experience with current ejection seat systems indicates that the exposure to low ambient temperature is of little significance as long as standard protective flight clothing is worn and items of equipment are neither damaged nor lost during escape. Surface survival in low ambient temperatures is discussed elsewhere.

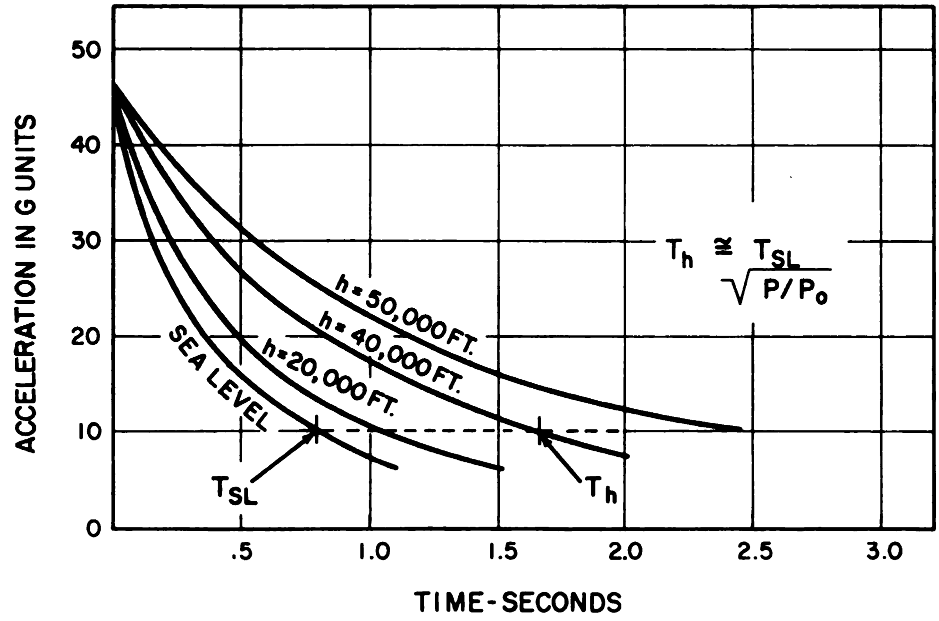


Figure 14–8. *Effect of altitude on deceleration time* (goodrich, 1956).

The high temperatures which can be caused by the ram rise effect at hypersonic speeds to date has not been experienced in emergency ejections. Aerodynamic heating is certainly a factor during the reentry phase of spacecraft operation. At high Mach numbers the temperature rise is very severe, approximately 75 times the square of the Mach number (for Fahrenheit scale). This is compounded by the low heat-exchange factor, which exists in the rarified higher altitudes.

Windblast tests, of large animals (chimpanzees), on high-speed rocket sleds, have produced severe third-degree burns on exposed body areas. These tests were conducted at a Mach number of 1.7 with a total windblast exposure of ten seconds (during acceleration and decay of the sled) and exposure of one second at peak velocity. Measured surface temperatures were 300° to 320° F, which would in itself not account for the injuries. However, the total transfer of heat due to the high airstream velocity is ascribed as the added causal factor.

It is doubtful that current open ejection seat systems will be used in hypersonic vehicles. Rather, some form of closed escape module will be used to counter thermal and other detrimental factors.

Tumbling or Rotational Stress.—The head-over-heels tumbling which occurs in conventional ejection seat systems has been discussed in terms of windblast and wind drag deceleration. In addition, tumbling and spinning during free fall has been covered in regard to disorientation and nausea. The centrifugal forces developed by these gyrations are also of importance.

Walchner (1958) investigated dummy drops from 83,000 feet which indicated that the human body may develop a spin rate of 465 rpm. Weiss, Edelberg, Charland, and Rosenbaum (1954) utilized a spin table to investigate animal and human reactions in order to establish tolerance limits. These tests showed that with the center of rotation at the heart, unconsciousness occurred in humans in three to ten seconds at 160 rpm. Indications are that spin rates of more than 400 rpm are fatal to man.

It is believed that the fatal rate of 400 rpm can be attained during free fall. U.S. Navy accident experience does not indicate that this has been a major problem in emergency escape to date. This is attributed to the low rate of occurrence of high-altitude high-speed escape events.

Walchner (1958) reported actual tumbling and spinning experiences of parachutists with rates as high as 240 rpm. This would produce a radial acceleration force of approximately 37 G at eye level, capable of producing severe retinal or cerebral-vascular damage.

Sperry, Neilson, and Barash (1955), in reporting USAF experience obtained from high-altitude downward ejection tests, indicated that human subjects suffered extreme disorientation, nausea, and vomiting during descent from 45,000-foot altitudes. Mazza (1951) reported similar results, including blurring of vision caused by the spinning of drogue-stabilized ejection seats during descent from 40,000 feet.

Spinning and tumbling will cause a combination of positive and negative accelerations whose effects will vary with the location of the center of rotation. When the heart is the rotational center, cardiodynamic and general circulatory effects are maximal. Animal studies have shown that at 150 rpm, with the heart the center of rotation, the A–V pressure difference and pulse pressure are reduced to less than five millimeters of mercury and cardiac output is nil. Tissue anoxia results and cerebral hemorrhage from damaged vascular walls can occur as spinning ceases and very high systolic blood pressure overshooting occurs. Circulatory impairment is not serious in humans at 125 rpm.

Hydraulic effects are greatest at the regions which are farthest from the center of rotation. When the center of rotation is located at the lower part of the body, conjunctival hemorrhage, periorbital edema, and hemorrhage into the sinuses and middle ear may occur. The thresholds of petechial hemorrhages of the conjunctiva have been determined. With the center of rotation at the iliac crest, the values vary from three seconds at 90 rpm to two minutes at 50 rpm. With the center at the heart, the ranges are ten minutes at 45 rpm to four seconds at 120 rpm.

Ejection Accident Summaries

Figures 14–9 and 14–10 show mean altitude at ejection and mean ejection airspeed, respectively. It is interesting to note that as aircraft performance (both altitude and airspeed) has increased, ejection altitude and airspeed have decreased.

These data are exact. However, all accident data are usually subject to several sources of bias during the chain of collection, reporting, interpretation, encoding, and analysis. Extrapolations from such data are hazardous since most causal factors of both accidents and injuries in accidents are multifactorial.

Additionally, changes are made every year in aircraft systems, ejection seats, and restraint systems. Each change can have a marked effect on the escape statistics, so long-term compilations, such as shown in table 14–7, must be viewed with great caution.

SPECIAL ESCAPE PROBLEMS

Previous discussion has largely centered about escape from fixed-wing conventional and jet aircraft. Additional problems are encountered with escape from rotary-winged aircraft and from submerged aircraft.

Rotary-Winged Aircraft

Helicopters have unique problems of escape. If engine failure should occur above a minimum altitude (usually 400 feet), there is no danger because the pilot can initiate autorotation and make a safe descent. If, on the other hand, engine failure occurs below a maximum altitude (usually 30–40 feet), the helicopter can land without killing anyone even if it drops like a stone. The "danger altitude," therefore is roughly below 400 feet and above 40 feet

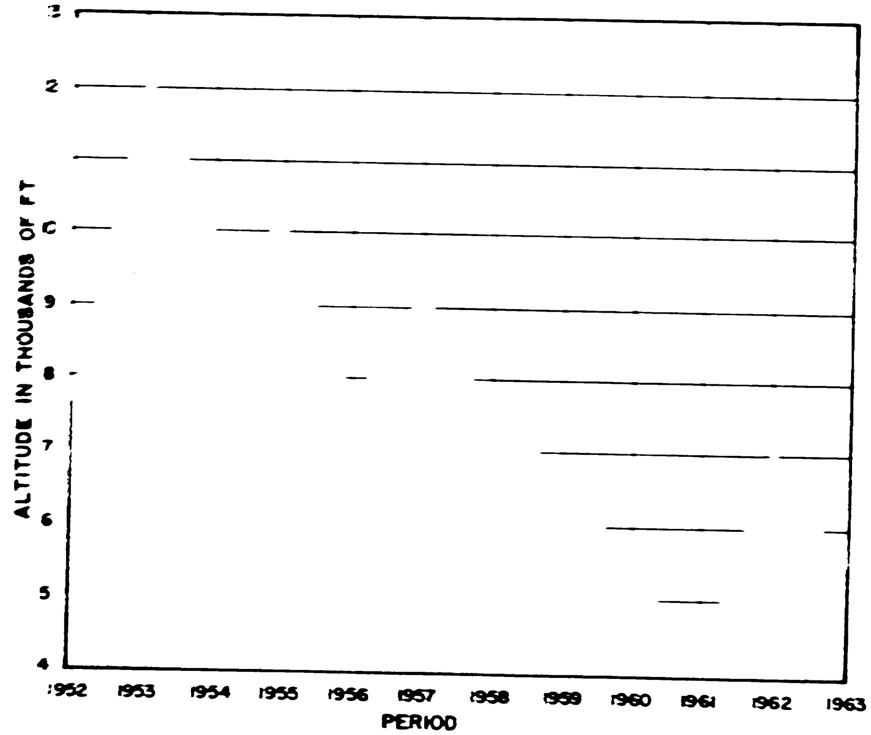


Figure 14-9. *Mean altitude at ejection by years* (naval aviation safety center, 1963).

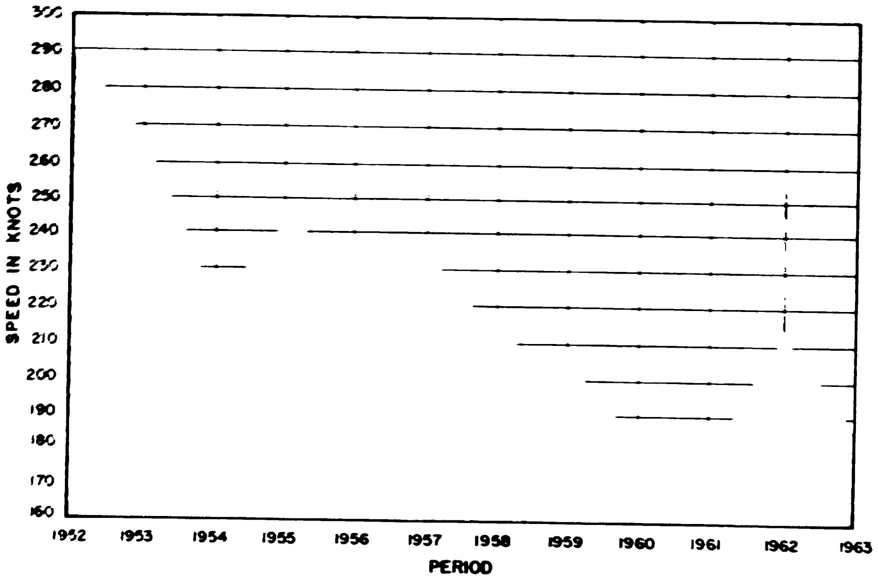


Figure 14-10. *Mean ejection airspeed by years* (naval aviation safety center, 1963).

Table 14–7. *Cumulative Injuries Resulting from Ejection According to Model 1949–1963*

Model Aircraft	Total	Resulting Injury to Personnel									
		Fatal		Critical		Severe/ Serious		Moderate/ Minor		None	
		No.	%	No.	%	No.	%	No.	%	No.	%
F–1	135	17	12			9	7	70	52	39	29
F–2	55	7	13			12	22	29	52	7	13
F–3	85	11	13			19	22	38	45	17	20
F–4	29	6	21			3	10	11	38	9	31
F–6A	74	10	14			11	15	40	54	13	17
F–8	196	28	14	1	1	22	11	92	47	53	27
F–9(TF9J)	284	66	23	4	1	52	18	134	40	28	10
F–11A	44	5	11			6	14	25	57	8	18
A–4	132	19	14			13	10	69	52	31	24
A–5	4	2	50					1	25	1	25
T–1A	8	1	12	1	12	1	13	3	38	2	25
T–2	18	1	5			5	28	5	28	7	39
T–33	55	7	13			8	14	33	60	7	13
	1					1	100				
	25	6	24	1	4	3	12	9	36	6	24
	1							1	100		
A–6A	1	1	100								
Totals	1,147	187	16	7	1	165	14	560	49	228	20

From *Emergency Airborne Escape Summary—CY 1963*, Naval Aviation Safety Center, Aeromedical Department.

for *single-engine* helicopters. Within this altitude zone, engine failure would result in severe injury or death to the aircrew. Naturally, these altitudes vary with the characteristics of the aircraft concerned, and are of little concern in twin-engine craft, since one engine can usually carry the load and prevent an accident.

An inflight fire, damage or loss of main or tail rotor, engine failure (or fuel starvation) at night, or unknown or unsuitable terrain for landing are occurrences which require emergency escape prior to landing. Currently, helicopter crews are provided with conventional personnel parachutes. However, their use has been minimal (only 13 bailouts in five years in all naval and Marine Corps helicopters) due to the low altitude at which escape situations usually occur (below minimum parachute recovery altitude), and the danger of impacting main rotor blades after egress (if helicopter is tumbling or inverted).

Proposed methods of extricating helicopter personnel have included: the installation of canted ejection seats which would eject the occupant out to the side, or the adaptation of a "chop-recovery" system which would incorporate linear-shaped charges to sever the main rotor and nonessential sections of the craft, and a recovery parachute system which would lower the occupied portion.

A recent parachute development, the ultraprecision parachute (UPP), formerly called the ultrafast-opening parachute (UFOP), appears to offer one solution to the low-altitude parachute deployment problem. This system does not rely on the inflow of air for deployment of canopy opening. Metal slugs sewn in the skirt of the parachute are ballistically fired radially from a deployment "gun." The parachute is thereby forcibly and rapidly opened. Tests show that successful deployment and landing can be made from an altitude of approximately 15 feet at zero velocity. At present, however, all these proposed solutions are strictly experimental, and in most cases, the crew must ride the aircraft in.

Underwater Escape

In naval air operations, water crashes and ditchings constitute a relatively high proportion of all major aircraft accidents at sea. Experiments (i.e., with engine shut down and aircraft simply dropped from flight deck height rather than having any greater velocity) have shown that jet aircraft float for a maximum of 60 seconds and then sink at a rate of up to several hundred feet per minute. Escape above water may not be possible (Ewing, 1966b).

Escape from a ditched aircraft may be accomplished as it sinks, or after it sinks, or the pilot may be unable to escape and his aircraft may come to rest on the bottom, or perhaps inverted, in relatively shallow water. Considerable time may elapse before rescuers can obtain scuba gear, locate the aircraft, and effect the rescue. During this period the aviator can continue to breathe 100 percent oxygen from the aircraft converter even though submerged. It must be clearly understood that use of oxygen-breathing capabilities in the crash situation is no substitute for escape attempts, but only a means of prolonging life while escape and/or rescue can be accomplished.

Oxygen Supply Under Water.—The nominal oxygen equipment installed in first-line jet aircraft, consisting of LOX converter, minireg, A-13A mask, and emergency bailout oxygen bottle, will continue to supply 100 percent oxygen even while submerged. The supply pressure will be equal to that exerted by the water at the regulator. Thus the nominal oxygen system constitutes scuba equipment. Since the mask-mounted minireg is mounted at an optimal position, breathing is possible whether the aircraft is upright or inverted, nose up or nose down, or lying on its side. The regulator is supplying oxygen at the ambient water pressure, there is no pressure differential, and therefore, no great inspiratory pressures are required.

Maximum time duration for underwater breathing naturally will vary with the oxygen supply available at the time of the accident, the development of oxygen toxicity, the depth, and the amount of exertion of the aviator. The first two factors noted are the most important. If the accident occurs with a full converter, the ten-liter converter is capable of 109 minutes of operation at 20 liters per minute average flow at a 100-foot depth. However, oxygen toxicity (see chapter 11) would develop within a few minutes at this depth, so the true limiting factor on duration of underwater breathing is the time required to develop oxygen toxicity, as noted under *Physiological Problems.*

When the aviator escapes from the aircraft or is rescued, he can disconnect himself from the aircraft oxygen converter and continue breathing uninterruptedly and easily from the bailout bottle and minireg during ascent to the surface.

The same is true of underwater breathing in the full pressure suit, because it, too, is attached to a bailout bottle. Since the helmet-mounted regulator provides oxygen at the same pressure as the surrounding water, the only differential pressure across the visor would be the one-inch water pressure differential normally exerted to prevent suit leakage into the breathing compartment. Specifically, breathing at simulated depths of 100 feet has been accomplished without difficulty.

Moreover, the full pressure suit itself is a reservoir of oxygen, in addition to the bailout bottle and aircraft LOX converter. If neither of the latter is available, or if both become exhausted, the aviator may, as a last resort, breathe from the body compartment of the suit by "slipping" the face seal. Tests at the Naval Aerospace Crew Equipment Department (ACED) have shown that breathing can be continued in this way in the upright position for about ten minutes, in the side position for about six minutes, but only a minute or so in the inverted position. These tests, it should be noted, were performed at a depth of only eleven feet.

In the single case of breathing from the suit, the major limiting factor on duration is carbon dioxide buildup with consequent hyperventilation. This is expected, of course, since rebreathing from the suit was not a design feature.

When the diluter-demand regulator is being utilized, the 100-percent-oxygen setting must be selected and the mask must be tightly adjusted to the face. For this reason, aviators using diluter-demand regulators should select 100 percent oxygen for all landings and takeoffs, since the system will work as well in mud or swamp as it does in seawater or firefighter's foam.

Regulator Location and Oxygen Supply Pressure.—Work performed at the Navy's Aerospace Crew Equipment Department has shown that one major factor in underwater breathing from aircraft oxygen supplies is the location of the regulator itself, since the pressure-sensing device which determines the pressure with which the oxygen is supplied is contained therein. It was found that if the regulator were mounted six inches below the suprasternal notch, the pressure on the lungs would be excessive. If the subject, with the regulator mounted at the same position, became inverted, the oxygen supply pressure would be so low that water would be inhaled. These experimental findings account in part for the location of the FPS regulator on the headpiece at the level of the mouth, and for the minireg on the A-13A at about the level of the suprasternal notch. A one-foot depth separation in water will result in approximately 0.5 psi (or 22mm Hg) differential pressure.

The regulator's position ideally should be on the line from axilla to axilla. Console-mounted regulators, usually of the diluter-demand type, are invariably below this level, thus causing the oxygen pressure to be positive. When breathing under water from them, the body should be bent toward the regulator so as to have the chest and the regulator at the same level, thus minimizing differential pressure.

Physiological Problems.—Residence underwater presents several potentially hazardous problems: CO_2 intoxication, N_2 intoxication, and O_2 intoxication.

Actually, all metabolic carbon dioxide is exhausted via the exhalation valve and, therefore, carbon dioxide intoxication presents no problem. Since 100 percent oxygen is the only inspired gas, nitrogen is being washed out constantly, and, therefore, neither is nitrogen intoxication a problem. Oxygen toxicity, however, very definitely is a problem and the other limiting factor, after O_2 quantity, on underwater breathing of 100 percent oxygen from the LOX converter and minireg.

Chapter 11 details the toxic effects of oxygen breathed at partial pressures in excess of 760mm Hg. The convulsions of oxygen toxicity may cause death by drowning if the mask is displaced during the convulsion.

Table 14–8 from the *U.S. Navy Diving Manual* shows the safe durations at the noted depths while breathing 100 percent oxygen. This information gives rescuers of an aviator trapped in his aircraft in shallow water some idea of the timeframe within which they must work.

TABLE 14–8. *Oxygen Depth—Time Limits*

[Depth and time limits of exposure when breathing pure oxygen during working dives]

1. Normal oxygen limits

Depth (feet)	Times (minutes)
10	240
15	150
20	110
25	75

2. Limits for exceptional operations

Depth (feet)	Time (minutes)
30	45
35	25
40	10

3. Emergency limits. See figure 14–11.

(*U.S. Navy Diving Manual*, July 1963)

Emergency limits are noted in figure 14–11. When the time limits noted are exceeded, grand mal convulsions can be expected. If the O_2 partial pressure is not decreased after the onset of the convulsions, status epilepticus will occur, with death the expected outcome (Beckman, 1967). Death from drowning *during* the convulsion must be anticipated and prevented, and rescuers should realize that convulsions, if they occur, are probably due to oxygen toxicity rather than to injury or other cause. It should be clearly understood that the noted time limits apply only to the aviator (or anyone else) breathing 100 percent oxygen, since rescue divers probably would be breathing air. If, however, the rescuers are using 100 percent oxygen apparatus, it applies to them also.

Figure 14-11 presents the results of experimental exposures to pure oxygen at various depths for various times, thus allowing estimation of the probabilities of development of oxygen toxicity. The proximity of possible warning symptoms and even convulsions to the Important Operation Limit Curve is apparent. Less apparent is the contrast between the perfect conditions that existed during these exposures and the conditions that would probably exist in the field. The experiments were conducted in a pressure tank. The work rates were moderate and uniform. The inspired gas was free of carbon dioxide. Two tenders were standing by each subject. It is likely that exposure to oxygen at these depths for the same times under operating conditions following a crash would produce a much larger proportion of unfavorable effects.

To delay the onset of oxygen toxicity, the aviator should remain perfectly still during rescue operations, once the rescuers know positively he is alive.

Physiological Problems During Ascent.—The major problem during ascent is caused by intrapulmonary gas expansion. If an individual ascends to the surface from 100 feet (or 4 atmospheres), the gas within his lungs will expand to four times its original volume. If not allowed to escape, it will rupture the alveoli and thus cause aeroembolism to the left heart and thence through the aorta. Such emboli can be highly variable in effects, since the consequences depend upon the area or organ where the emboli eventually block the arteries. They are frequently fatal.

If the breath is held during ascent, a change in depth of only a few feet may be fatal. Under these circumstances, death due to aeroembolism has occurred in ascent from depths of only 15 feet. At least one such case has followed ascent from the bottom of a swimming pool, as reported in the *Navy Diving Manual.*

On the other hand, safe ascents can be made from depths of more than 100 feet without any breathing appliance, providing the individual exhales continuously during his ascent. This is called the "Blow-and-Go" method of ascent and is the one to be utilized by aviators when escaping from aircraft underwater, where no bailout oxygen is available. As noted earlier, continuous respiration utilizing the bailout oxygen bottle on ascent also will equalize pressure and prevent gas expansion damage to the lungs.

Underwater Ejection.—Most NATOPS procedures call for canopy jettison when a ditching or water crash is imminent. If the canopy is successfully

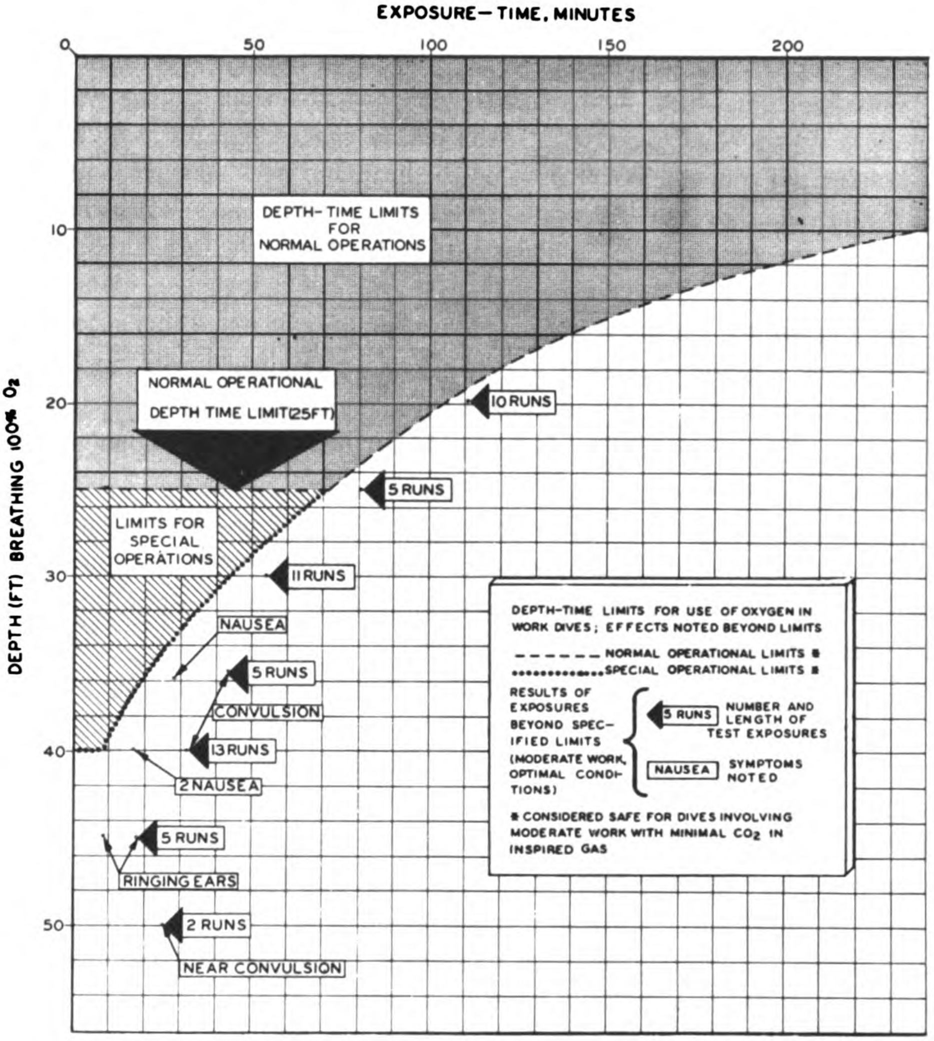


FIGURE 14–11. *Oxygen depth-time limits* (U.S. Navy Diving Manual, 1963).

removed prior to water impact, escape is not too complicated. However, in many instances, the canopy-jettison mechanism is designed to lift the leading edge of the canopy sufficiently that normal windblast will complete the job of removal. In the slow-velocity water crash, typified by the "cold" catapult shot, windblast may not be sufficient to complete the canopy removal sequence. In other cases, there is simply no time available to jettison the canopy. Also, underwater canopy jettison is usually unsatisfactory. If the canopy cannot be removed, and if through-the-canopy ejection is possible in the particular aircraft type, it may be attempted as a "last ditch" method of escape, especially in an aircraft rapidly descending in deep water.

Previous studies have shown that the canopies of various aircraft will implode at depths of 15 to 40 feet. If this occurs, canopy removal is no longer a problem, of course. Water drag on the head and neck, as well as impact of the head on the canopy,may cause severe or even fatal head and neck injury. To lessen the risk of injury, the seat must be fired with the face curtain over the head.

Having pointed out the severe hazards, it can now be stated that successful underwater ejection is possible and has been accomplished with no injury on several occasions. Again, it must be emphasized that underwater ejection is a hazardous undertaking which should be used only if no other means of escape is possible.

The comments concerning underwater ejection may or may not apply to rocket-type seats, since these have not been adequately tested. Any such use of a rocket seat should be considered extremely hazardous at least until testing has been accomplished.

TRAINING

Navy policy does not require aviation personnel to perform actual parachute jumps. Aircrew personnel are required to experience certain functions which occur during emergency escape in order to learn the proper techniques and procedures without exposing individuals to unwarranted hazard. The bailout and ditching drills previously discussed are one type of training. In addition, the following types of training are available.

Dilbert Dunker

This device consists of a simulated cockpit section mounted on rails which extend into a swimming pool. The trainee, after receiving proper indoctrination, is seated in the cockpit with shoulder harness and lap belt secured. The cockpit assembly is released and slides into the water and the forward section (nose) is rotated down until the cockpit is inverted and completely immersed. After all motion stops, the trainee releases himself, pulls himself clear, and surfaces.

Safety precautions are extensive. Normally, specially trained scuba-equipped swimmers are located in close proximity. They observe the actions of the trainee and lend assistance if necessary.

Harness Drop

The trainee dons parachute harness identical to that which he wears in flight. From a platform located above a swimming pool he is attached to an apparatus which suspends him over the surface of the water. A release mechanism causes the trainee to drop (eight to ten feet) into the water, simulating actual parachute landing forces. The trainee then releases himself from his parachute harness.

At some facilities the lifejacket is then inflated, the pararaft inflated, and the trainee demonstrates his ability to board the raft in the prescribed manner.

Water Drag

While standing on the edge of a swimming pool, the trainee attaches his parachute harness to a cable which is in turn attached to an electric hoist. On signal, the trainee jumps into the water. The hoist is started and the cable reeled in causing the trainee to be dragged through the water simulating a parachute dragging a man under light wind conditions. The trainee rolls on his back and performs the appropriate release tasks.

At some bases where there is no such swimming pool facility, open water training is conducted using a small boat to simulate dragging conditions.

Helicopter Hoist

The trainee is located in the water (swimming pool). Each of the types of hoisting apparatus in current use (fluted seat or sling) is placed in the water. The trainee demonstrates his ability to board or enter each device properly and is hoisted vertically just clear of the water.

Many squadrons utilize "live" helicopter hoisting as a means of training. Arrangements are made with a helicopter squadron or unit to perform actual hoists from an offshore location or from a nearby bay or lake. This permits indoctrination in boarding both the rescue device as well as the rescue craft while in the actual downwash created by the helicopter rotor blades.

In the "live" situation trainees also can ignite night and day signal flares to learn proper ignition procedures. These training exercises should be conducted while wearing antiexposure suits whenever possible.

Sea Survival

Recent events, in combination with a long-felt need, have caused adoption of a new training program in sea survival at the Naval Air Basic Training Command. A specially-equipped LCM is utilized along with two monitor boats.

In operation, the boats are taken into the Gulf of Mexico, and each trainee dons a parachute harness. This is attached to risers which are attached to a cable. Rails attached to the upper deck of the LCM at a height of 25 feet above the water are used to propel the trainee over the fantail of the LCM and thus clear of the screws. When the end of the rails is reached, a tripping device drops the trainee into the water and he is then dragged through the water at ten knots in an extremely realistic simulation of actual conditions. He is taught to use the risers to flip himself on his back, to free up his PK II liferaft, to release himself, and then to inflate the raft and board it.

The forward velocity of the LCM is such that the trainees are deposited at widely separated locations where they are permitted to remain afloat in the raft for about four hours, utilizing the solar still to obtain water, firing flares, and using other signalling devices, and experiencing the mild exposure incident to the particular time of year.

Each step is carefully observed by personnel aboard the monitor boats; scuba-equipped swimmers are on hand to aid any trainee whose situation becomes hazardous.

Bailout and Ditching Drills

Bailout and ditching drills are required in most commands. These are typically initiated either immediately after crewmembers have embarked and are strapped in, or immediately following aircraft parking and engine shutdown. Crews are scored on a basis of following correct procedures and exiting the aircraft within the prescribed time. Conscientious crews are able to correct many faulty procedures and to shorten abandon time appreciably. Records are normally maintained by the squadron safety officer or the aircraft commander to insure that drills are held periodically and that exit times are reasonable or improving.

These drills are most effective when no forewarning has been given crewmembers. At some activities the squadron safety officer or his representative will meet an arriving aircraft and give a prearranged signal to the pilot who in turn will announce either "bailout drill" or "ditching drill" to the crew. The total elapsed time is then recorded and errors in procedures noted. This permits immediate discussion of problem areas and also serves as an indication of errors common to all crews (which may be due to incorrect lecture content).

Ejection Seat Training

In addition to the training discussed under conventional bailout, pilots of ejection seat-equipped aircraft receive training regarding the ejection seat per se.

Procedures Trainer.—Procedures trainers are available for many high-performance aircraft. These are static devices which duplicate the specific seat installed in a particular aircraft and, in some cases, a portion of the cockpit itself. Some operational flight trainers (OFTs) can be used as ejection procedures trainers.

The procedures trainer provides indoctrination in the sequence of activities necessary for successful escape, body positioning, actuation sequence, secondary methods of actuation, and the like. In addition, ground emergency escape (nonejection seat) procedures can be practiced.

Static Seat.—While afloat or when stationed at a place which does not have a procedures trainer, a deactivated ejection seat may be used as a training device. Extreme care must be taken to insure that the seat has truly been made safe. All cartridges, both propulsion and gas initiator, must be removed and should be actually sighted by personnel prior to sitting or actuating seat controls. This is best accomplished during required periodic inspection of aircraft and seat when it will not interfere with aircraft utilization.

Ejection Trainer.—Ejection seat trainers which provide aviators with dynamic ejection seat training are available at many air stations.

The trainer consists of an ejection seat, propelling charge, and a set of rails which project upward and backward at an angle of 18 degrees from the vertical. The airman performs the same preejection and ejection tasks required by an actual ejection seat system. Upon actuation of the face curtain, a small catapult charge is fired and the seat and occupant are propelled up the guide rails. A means is provided for lowering the occupied seat back down to ground level. This type of training has proved to be extremely effective. Pilots who have made emergency ejections indicate that the dynamic training prepared them for the actual emergency condition and aided in relieving apprehension concerning catapult firing.

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CHAPTER 15

Selection of Aviation Personnel

PSYCHOLOGICAL SELECTION

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PHYSICAL SELECTION

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PSYCHOLOGICAL SELECTION

ROSALIE K. AMBLER
NAVAL AEROSPACE MEDICAL INSTITUTE

INTRODUCTION

The selection, or screening, process for the U.S. Navy and Marine Corps aviation program strives to procure and retain personnel who are physically fit and temperamentally adapted to aviation and to military life. The process is in three steps:

1. Officer and enlisted procurement.
2. Psychological testing.
3. Aviation physical examination.

Officer and enlisted procurement procedures are under the Bureau of Naval Personnel (also Marine Corps Headquarters or Coast Guard) and are not a typical responsibility of the Flight Surgeon. This chapter will not elaborate on this area other than to indicate that it includes a review of the candidate's application by a selection board, personal reference checks, a personality rating by a senior officer, and a National Agency (Security) Check. Selection on the basis of psychological and physical examinations is under the Bureau of Medicine and Surgery and, therefore, is a direct responsibility of the Flight Surgeon. The practices regarding these examinations are the primary concern of this chapter.

Since two bureaus function simultaneously in implementing the screening process, the Flight Surgeon will frequently find himself working with procurement officers. In addition to completing step 1 above the procurement officer will usually be under pressure to fill his assigned quota of recruits. When the Flight Surgeon, in implementing the psychological and physical examinations, rejects an applicant, this may be perceived as a barrier to quota fulfillment. The Flight Surgeon must therefore exert every effort to establish a cooperative and effective working relationship with procurement personnel. The best way to prepare for this role is to be informed thoroughly on all aspects of the psychological and physical examinations for aviation.

The term "selection" as it applies to Flight Surgeon duties is used in this chapter in its broadest sense. This usage includes the initial selection of aviation candidates as well as the removal from flying status of those who develop disqualifying defects. This broad usage is most applicable to the aviation physical examination.

Aviation selection practices have been standardized so that consistent results may be achieved regardless of where or by whom the practices are applied. It is the purpose of this chapter to present an overview of aviation selection practices as they relate to the Flight Surgeon and to provide reference sources for the technical details required in the consistent application of the practices.

PSYCHOLOGICAL TESTING

The U.S. Navy and Marine Corps Aviation Selection Test battery consists of four tests covering a wide range of abilities, attitudes, interests, and personal characteristics. The tests were constructed specifically for naval aviation to provide a procedure which:

1. Gives an accurate probability statement of a candidate's potential for completing aviation training.
2. Is economical and does not take much time.
3. Permits standardized evaluation of thousands of candidates annually throughout the U.S.A. and the Fleet.

Completely infallible selection procedures for any sizable training program, either in or out of the military, are theoretically impossible. Some of the candidates who pass these tests will fail training, and some who fail the tests might have passed training. Extensive research has shown, however, that the better the candidate does on the tests, the more favorable are the odds that he will complete training. Conversely, the odds are against completion for those with low scores. Short of giving actual time in a training aircraft, the Navy and Marine Corps Aviation Selection Tests are the best means available for assessing the candidate's chances of completing training *before* he actually enters training.

Once the candidate enters training, data from his performance in training are added to the selection test data, and, by means of the computerized Pensacola system of quality control, progressively more precise probability statements of subsequent performance in training and the Fleet can be made. These probability statements aid administrative officers in decisions about the retention or failure of any given student experiencing difficulties in training. This quality control system may be described as a *secondary* selection process as opposed to the *primary* selection process of the paper and pencil psychological tests. Implementation of the primary selection process is the direct concern of the Flight Surgeon in the field. The secondary selection process is more the concern of Training Command personnel in cooperation with research personnel of the Naval Aerospace Medical Institute.

Development

The U.S. Navy and Marine Corps Aviation Selection Test battery was developed from extensive field studies on many tests. Each test was given experimentally to a large number of applicants. The responses were kept in a closed file until they could be correlated with the ultimate success or failure of the men involved. Test material was retained for further investigation if the results showed:

1. Statistically significant differentiation between successes and failures in training.
2. Significant augmentation of the predictive value of tests then in use.

Cross-validation studies on additional large groups of flight students produced an optimal combination of tests which satisfied the above conditions and which permitted efficient standardization of procedures for country- and Fleetwide usage.

Monitoring of the tests is carried on continuously to insure that the tests are performing as intended. Research and development of future revisions of the battery are commenced simultaneously with the implementation of any new battery in the field.

Description

Table 15–1 contains a descriptive schema for the present battery. Part I, or the Aviation Qualification Test (AQT), is considered an academic screen. Individuals who fail this test are not considered good risks to complete the academic portion of training or to function as professional military officers. The correlation between ground grades and the AQT score is usually of the order of .60 uncorrected for sample truncation. An applicant must pass this test to be considered for further processing.

Since qualifying scores vary for different aviation specialties, cutting scores will not be discussed in this text. The reader will find references at the end of this section for detailed technical information about the tests and their use.

The Flight Aptitude Rating (FAR) is derived from a composite of the three tests of Part II. Research has shown that each of these tests makes a discrete contribution to prediction of the criterion of pass versus separation from actual flight training. Correlations of the FAR with the pass/fail flight dichotomy are generally of the order of .30 uncorrected for sample truncation. At the present time, sample truncation is considerable since about 32 percent of the candidates are rejected on the basis of the FAR and seven percent to ten percent on the AQT.

TABLE 15–1. *Description of Aviation Selection Tests*

Test Title	Length	Item Content
PART I: Aviation Qualification Test (AQT).	115 items, 60 min	Quantitative ability. Verbal ability. Practical judgment. Clerical speed and accuracy. Direction following.
PART II:		
Mechanical Comprehension Test (MCT).	76 items, 45 min	Mechanical aptitude.
Spatial Apperception Test (SAT)	30 items, 10 min	Spatial orientation.
Biographical Inventory (BI)	120 items, untimed	Evidence of early maturity. Early risk-taking behavior. Informal acquisition of aerospace knowledge. Selected personal history items related to aviation success. Selected attitudes and interest items related to aviation success.

Effectiveness

Figure 15–1 contains a schema showing the probability of completing pilot training for various combinations of the FAR and AQT. The scores represented are converted scores which range from one to nine. Information about derivation of these conversions can be found in the manual cited in the *Reference Materials* paragraph. Figure 15–1 does not contain any scores of one or two because no pilot trainees have been admitted recently with scores this low. It is estimated, based on experimental data for the current battery, obtained before it was implemented in the field, that approximately 75 percent of those scoring one or two on the FAR would fail, and that those who passed would be pilots of borderline quality in the Fleet.

Table 15–2 contains estimates of the training separations to be expected for various selection standards. Table 15–3 compares these standards in terms of current dollar savings.

Standards

The Bureau of Medicine and Surgery sets the minimum acceptable grades on the selection tests. The Bureau of Naval Personnel, the Coast Guard,

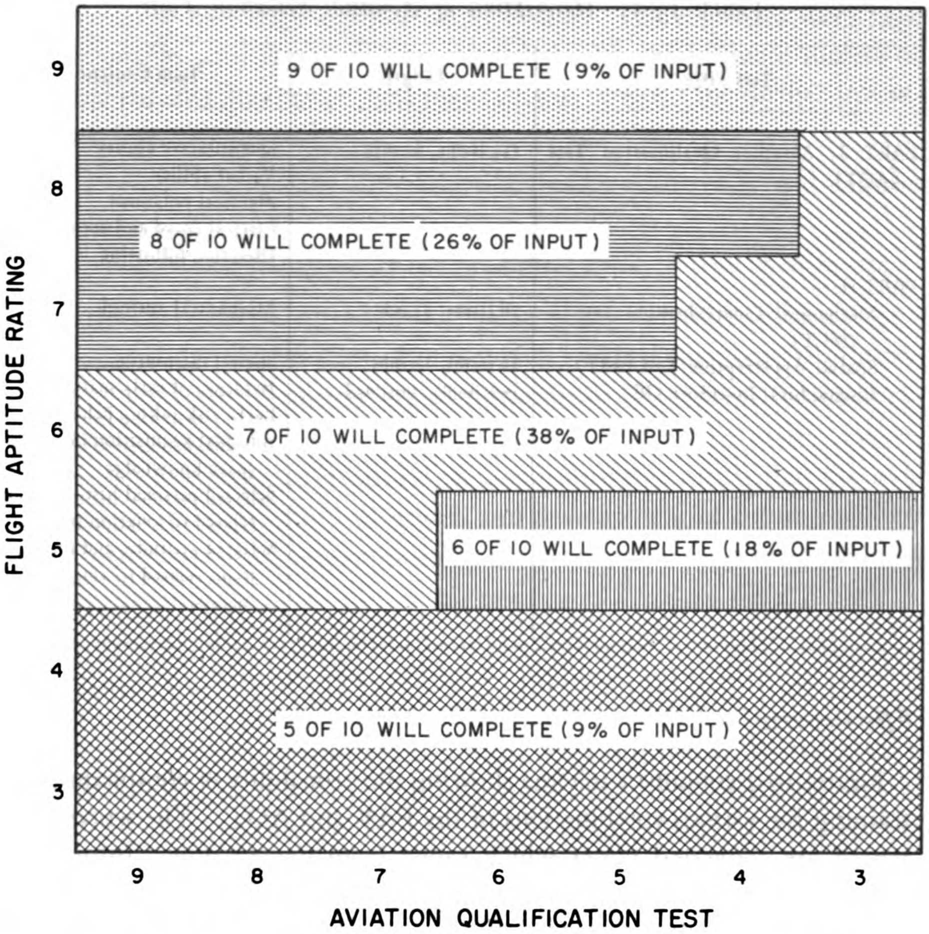

FIGURE 15–1. *Probability of completing naval aviation training for various combinations of AQT and FAR scores.*

or the Marine Corps may superimpose higher standards if the manpower supply and demand indicate that this is desirable for their respective services. Under no circumstances may minimums be set below the BUMED level.

Reference Materials

The Flight Surgeon who anticipates any involvement with the processing of flight applicants should become thoroughly familiar with:

Examiner's Manual and Scoring Instructions for the United States Navy and Marine Corps Aviation Selection Tests (*NAVMED 5098*).

TABLE 15-2. *Estimated Inputs and Training Separations for Various Selection Standards*

Minimum Standards	Input	Training Separations	Designated Aviators
No selection tests used	1,754	754	1,000
AQT-3 FAR-3	1,587	587	1,000
AQT-3 FAR-5	1,475	475	1,000

TABLE 15-3. *Comparative Savings per 1,000 Aviators for Various Selection Standards*

Comparison	Difference in Separations		Est. Cost per Separation		Savings
Between AQT-3/FAR-5 and no tests	279	×	$12,500	=	$3,487,500*
Between AQT-3/FAR-3 and no tests	167	×	$12,500	=	$2,087,500
Between AQT-3/FAR-5 and AQT-3/FAR-3	112	×	$12,500	=	$1,400,000

*For a 2,200-per-annum program this represents a savings of $7,672,500.

From time to time BUMED and BUPERS Instructions and/or Notices are issued regarding minimum scores and modifications in materials, procedures, and standards. The Flight Surgeon is advised to request the manual and current information in this area from:

Aviation Operational Psychology Branch
Bureau of Medicine and Surgery (Code 513)
Navy Department
Washington, D.C. 20390

PHYSICAL SELECTION

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Introduction

To the recently designated Flight Surgeon, the routine physical examination of flying personnel can be the most boring and exasperating of duty assignments in terms of monetary value (in the case of candidates for flight training) and in career implications for naval aviators. Nevertheless, the importance of a thoroughly performed and accurately recorded physical examination cannot be overemphasized.

The *Manual of the Medical Department* is the basic reference for this section. In keeping with the importance of this subject, *MMD* is quite detailed in almost every aspect, and all Flight Surgeons must thoroughly familiarize themselves with the entire contents of chapter 15. Only a bare outline is presented here.

THE AVIATION PHYSICAL EXAMINATION

The Aviation Physical Examination is *defined* as an examination conducted to determine whether or not a person is physically qualified and aeronautically adapted to engage in frequent aerial flights. The extent of the examination is determined by the character of the duty to be performed by the person who will make such flights. The *objective* of the Aviation Physical Examination is to select for aviation duty only those individuals who are physically and mentally qualified for such duty, and to remove from such duty those who may become temporarily or permanently unfit because of physical or mental defects. Further qualifications of this objective are contained in Paragraph 1559, *MMD.*

EXAMINER QUALIFICATIONS

The Aviation Physical Examination may be performed only by a Flight Surgeon or by an Aviation Medical Examiner who is on active duty and currently assigned to a Flight Surgeon or Aviation Medical Examiner billet or aviation activity authorized by the Chief of Naval Personnel or by proper authority of the Army or Air Force to conduct such examinations. Only medical officers who have successfully passed a course at a school of aviation medicine of the U.S. Armed Forces, leading to the designation of Aviation Medical Examiner or Flight Surgeon are so designated.

CLASSIFICATIONS AND PHYSICAL STANDARDS

There are several classifications of aviation personnel ranging from Naval Aviator and Naval Flight Surgeon to aircrewman. Each classification has its particular physical qualifications or physical standards, developed over many years of aviation experience, but which are constantly changing with the demands of new aircraft and new missions. There is a good, though perhaps not obvious, reason for *every* standard.

REQUIRED OCCASIONS FOR EXAMINATION

In addition to having different physical standards for the various classifications of aviation personnel, examinations for each classification must be conducted on occasions as specified in *MMD,* Chapter 15.

REQUIRED REPORTS

On certain occasions of examination, a report of the results must be made to the Bureau of Medicine and Surgery via Standard Forms (SF) 88 and 89. Although identical examinations may be performed on other occasions, the *report* of other such examinations *may* be limited to an entry on the SF 600 or the submission of a Clearance Notice (Aeromedical), NAVMED 1381, prior to returning to duty involving flying. Other report forms are delineated in detail in Chapter 15, *MMD.*

NOTES AND COMMENTS ON SELECTION OF AVIATION PERSONNEL BY AVIATION PHYSICAL EXAMINATIONS

The Flight Surgeon performing selection examinations on prospective candidates for aviation training or duties operates under many pressures. He may be overworked. His duty station may be a recruiting facility which has a quota for aviation candidates. He may be deeply impressed by the motivation of certain prospective candidates who will perform any task in their attempt to become a part of the aeronautical organization. He may detect abnormalities on the examination which he may consider to be temporary (or permanent), and the decisions regarding these must be made at his own discretion. Other persons, unknown to the Flight Surgeon, may give advice concerning the completion of the medical history form, and such actions may result in a completely misleading history. Misguided friends of the prospective candidate may advise him to tell little white lies on the Standard Form 89 (Report of Medical History) to enhance his chances of being found physically fit as a candidate for duty involving flying.

Such pressures are as old as aviation itself, and therefore current mechanisms for preventing unqualified personnel from becoming candidates are extremely refined and well worked out. The person who is not physically qualified for duty involving flying is inevitably detected before he enters flying training. Each candidate for this training must have a thorough and tough Aviation Physical Examination upon reporting to the Training Command. Any attempts by misguided friends or sympathetic physicians to "pass" such a person at an earlier examination elsewhere result only in detection of his defects at the Training Command. Upon detection, the person is removed from the program for which he is a candidate and is usually transferred elsewhere. The net result: wasted time, money, and effort, and a great disappointment to the prospective candidate.

The most careful history taking, systems review, and physical examination are of no value if these procedures are not accurately reported. The Flight Surgeon must fill out the rough report and carefully compare it with the final report presented for his inspection and signature. The aviation candidate deserves the benefit of the most careful examination and accurate reporting of which the Flight Surgeon is capable.

In the examination of designated Naval Aviators, Naval Flight Officers, and other expensively trained and experienced aviation personnel, there is a tendency to be more lax in applying physical standards than in the examination of candidates. The only laxity permissible is that contained in Chapter 15 of the *Manual of the Medical Department,* which defines the somewhat lower physical standards for retention.

A common error made by the inexperienced Flight Surgeon is that of glossing over or omitting mention of physical defects which, although "technically" considered disqualifying (i.e., are listed as such in *MMD*), are easily remediable and "cause no hazard to the individual nor to his ability to fly." Such action is a disservice to the aviator or to the candidate, because, in fact, conditions listed in *MMD* as being disqualifying *do* represent an added hazard to the pilot, either actually, as in flight, or potentially, as in a survival situation. If in doubt as to diagnosis or disposition, the Flight Surgeon should send the patient to the nearest Naval Hospital for consultation and indicated studies.

When the precise nature of the defect is known, corrective therapy can be arranged at a time convenient to the individual. If the defect is not remediable, it should be reported on the SF 88, and waiver recommended, if the Flight Surgeon determines that such defect will not hamper the individual in the performance of his duties, including flying. In this way, the Flight Surgeon can save time for both the aviator and himself. Correction of such defects is much more difficult and time-consuming in other places and at other times, particularly on a tour of sea duty.

Methods for detection of malingerers are covered elsewhere in this manual and in the *Manual of the Medical Department.*

Routine can become so well established that errors in examination, testing, or recording are perpetuated. The Flight Surgeon must be aware that an occasional technician will report normal results on routine examinations he has not actually performed. Such behavior on the part of any individual in the naval service is grounds for disciplinary action, just as it is grounds for legal action in civil life. Inspirational leadership by the Flight Surgeon, and treatment of offenders such as they would receive in civil life, will eliminate such chicanery, but the Flight Surgeon must be constantly alert. He should supervise the examination routine personally to insure that such problems do not arise.

CHAPTER 16

Physical Fitness

- NUTRITION
  - Carbohydrates
  - Fats
  - Proteins
- EXERCISE AND RECREATION
- SLEEP AND REST
- FATIGUE
  - The Physiological and Psychological Bases of Fatigue
  - Operational Factors Contributing to Fatigue
  - Identification of Excessive Fatigue
  - Control of Fatigue
- DONATION OF BLOOD
- MEDICATIONS
  - Antihistamines and Decongestants
  - Antibiotics
  - Tranquilizers
  - Antimalarial Drugs
  - Motion Sickness Drugs
- STIMULANTS
  - Central Nervous System Stimulants
  - Coffee
- SMOKING
- ALCOHOL
  - Effects of Alcohol on Man
  - Alcohol Hangover
  - Elimination of Alcohol
  - Treatment of Alcohol Problems
- REFERENCES

AN AVIATOR, like the aircraft he flies, requires preventive maintenance. One important, but often ignored, aspect of this maintenance is physical fitness. The airman is not required to perform heavy physical work in flight, but he must be physically fit to meet the combination of skilled perceptual-motor performance requirements, an alien environment, unusual stresses, and little tolerance for error which make up modern naval aviation. In addition, and most importantly, he must be prepared at all times to leave the civilized environment of the climate-controlled cockpit and, within a period of seconds, enter a savage world of jungle, open sea, mountains, arctic regions, or desert, where his physical fitness determines whether he survives or not. Many an aviator has died because he could not hold his breath that extra few seconds or walk that extra few miles, or quite reach the face curtain under the heavy accelerations of a doomed aircraft.

One does not "don" physical fitness along with the survival kit and flight suit. It must be present at all times, and this means constant awareness and lifelong effort to achieve and maintain an acceptable state of fitness. Nutrition, exercise and recreation, and sleep and rest are, in an otherwise healthy body, the most important determinants of physical fitness. Each is equally important.

Guidance for the development and maintenance of individual physical and mental fitness is provided in the OPNAV Instruction of the 3740.7 series entitled "Fitness of Flying Personnel."

NUTRITION

The Flight Surgeon is concerned with nutrition in three ways. These are:

1. *Education.* The Flight Surgeon should periodically instruct aviators in the fundamentals of good nutrition and in the importance of nutrition for flying fitness and general well-being.

2. *Consultation.* The Flight Surgeon should consult with and advise individuals in his unit who have nutrition problems of any kind.

3. *Supervision.* The Flight Surgeon should inspect and, if necessary, supervise meal planning, preparation, and facilities on land, on sea, and in the air. Particular attention should be given to problems of inflight feeding. To accomplish this supervision, the importance of carbohydrates, fats, proteins, vitamins, and minerals for the diet of aircrewmen must be understood.

Even more importantly, the *form* in which inflight meals are presented determines the success of the feeding in many instances. For example, it is unfortunately all too common to find that the flight lunch for an A–1 pilot on a long-duration, low-altitude mission contains an unpeeled orange, a can of tomato juice, or some other enticing and nutritious food which simply can't be eaten by a man who can only spare one hand for eating. The Flight Surgeon who is aware of the requirements of his unit can greatly aid in eliminating such maddening events by consulting with food service personnel. When special missions are laid on, part of the planning should involve the inflight feeding that will be most appetizing, convenient, and nutritious.

Daily allowances for critical nutritional elements recommended are presented in table 16–1.

TABLE 16–1. ***Recommended Daily Dietary Allowances for Men (allowances are intended for persons normally active in a temperate climate)***

Age years	Weight	Height	Calories	Protein	Calcium	Iron	Vitamin A Value	Thiamine	Riboflavin	Niacin Equiv.	Ascorbic Acid
from—to	Kg. (lb)	cm. (in)		gm	gm	mg	IU	mg	mg	mg	mg
18–35......	70(154)	175(69)	2,900	70	0.8	10	5,000	1.2	1.7	19	70
35–55......	70(154)	175(69)	2,600	70	0.8	10	5,000	1.0	1.6	17	70
55–75......	70(154)	175(69)	2,200	70	0.8	10	5,000	0.9	1.3	15	70

Adapted from Home and Garden Bulletin No. 72, U.S. Department of Agriculture, Sep. 1964.

Carbohydrates

The need for glucose varies greatly, making it difficult to recommend a daily requirement. The optimum amount of carbohydrates should be judged individually, based upon height, weight, amount of activity, and operational environment. Between 1,600 and 2,000 carbohydrate calories, or about 60 percent of the daily caloric intake, appears to be adequate for aviators. Principal sources of carbohydrates are sugar, starches, cereal grains, vegetables, and fruits.

Fats

Fats represent an important item in the diet of airmen. They are a concentrated source of energy, having an effective physiologic energy equivalent of about nine Kcal per gram ingested, or over twice that of carbohydrates or proteins. Fat deposits thus serve as an energy reservoir. However, since humans are rarely called upon to survive for long periods in the complete absence of food, the building of large fat deposits is undesirable for a number of reasons.

The most common sources of fats are fatty meats, butter, margarine, cream, cheeses, whole milk, shortenings, mayonnaise, salad dressings, and egg yolks.

Proteins

Proteins are of obvious dietary importance inasmuch as they are indispensable elements in the building of living material. Even though the supply of proteins is abundant, they cannot be used for body growth or maintenance purposes unless the diet provides a satisfactory quantity of carbohydrates and fats also. This is because the energy requirements of the body must first be satisfied. With restricted caloric intake, energy is provided through the breakdown of tissue elements, including protein. Research concerning this problem (Geiger, 1960) indicates that unless about 50 to 60 percent of the caloric requirement is satisfied by fat or carbohydrate, the amino acids cannot be used for protein synthesis or other specific purposes. This leads to the recommendation that proteins not be included in any emergency survival rations that provide a daily quota of 900 calories or less.

The best sources of proteins are meat, fish, soybeans, poultry, eggs, milk, and cheese. Although it has been shown that man can live on as little as 45 grams of protein daily (Davidson & Passmore, 1963), provisions for emergency exercise and a varied diet require more protein than this. The recommended intake for a man of average size, who is moderately active, is one gram of protein per kilogram of body weight daily, or about 15 percent of the daily caloric intake.

Table 16–2 presents information concerning carbohydrates, fats, proteins, and calories of selected foods.

Table 16–3 presents minimum daily requirements for vitamins and essential mineral elements.

TABLE 16–2. *Carbohydrate, Fat, Protein, and Calorie Contents of Selected Foods*

Item	Measure	Protein (gm)	Fat (Total Lipid) (gm)	Carbohydrate (gm)	Calories
Cow's milk, whole	1 cup	9	9	12	160
Milk, nonfat (skim)	1 cup	9	tr	13	90
Light cream	1 cup	7	49	10	505
Heavy cream	1 cup	5	89	7	840
Cheddar or American cheese	1 in. cube	4	5	tr	70
Creamed cottage cheese	1 cup	31	9	7	240
Cream cheese	1 oz	2	11	1	105
Cocoa	1 cup	9	11	26	235
Ice cream	⅛ qt./brick	3	9	15	145
Ice milk	1 cup	9	10	42	285
Boiled egg	2 eggs	13	12	1	160
Scrambled eggs w/milk & fat	1 egg	7	8	1	110
Bacon, broiled or fried, crisp	2 slices	5	8	1	100
Hamburger-lean, broiled	3 oz	21	17	0	245
Roast beef, lean & fat	3 oz	17	34	0	375
Sirloin steak, broiled	3 oz	20	27	0	330
Round steak, broiled	3 oz	24	13	0	220
Canned corned beef	3 oz	22	10	0	185
Beef & vegetable stew	1 cup	15	10	15	210
Chicken, broiled	3 oz. w/out bone	20	3	0	115
Chicken, breast, fried	2.7 oz	25	5	1	155
Drumstick, fried	2.1 oz	12	4	tr	90
Canned chile con carne w/beans	1 cup	19	15	30	335
Lamb chop, broiled	1 chop, 4.8 oz	25	33	0	400
Roast ham, lean & fat	3 oz	18	19	0	245
Pork chop	1 chop, 3.5 oz	16	21	0	260
Roast pork	3 oz	21	24	0	310
Bologna, slice 4.1 x 0.1 in	8 slices	27	62	2	690
Frankfurter, cooked	1 frankfurter	6	14	1	155
Veal cutlet, broiled	3 oz	23	9	9	185
Bluefish, baked or broiled	3 oz	22	4	0	135
Crabmeat	3 oz	15	2	1	85
Fish sticks, breaded	8 oz. pkg	38	20	15	400
Haddock, fried	3 oz	17	5	5	140
Shrimp, canned	3 oz	21	1	1	100
Swordfish, broiled w/butter or margarine.	3 oz	24	5	0	150
Tuna, canned in oil, drained, solids	3 oz	24	7	0	170
Red beans, dry	1 cup	15	1	42	230
Lima beans, cooked	1 cup	16	1	48	260
Cashew nuts, roasted	1 cup	23	62	40	760
Dried coconut, sweetened	1 cup	2	24	33	340
Peanut butter	1 tbsp	4	8	3	95
Peas, split, dried, cooked	1 cup	20	1	52	290

TABLE 16–2. *Carbohydrate, Fat, Protein, and Calorie Contents of Selected Foods*—Con.

Item	Measure	Protein (gm)	Fat (Total Lipid) (gm)	Carbohydrate (gm)	Calories
Asparagus, canned	6 spears	2	tr	3	20
Beets, cooked, diced	1 cup	2	tr	12	50
Brussels sprouts	1 cup	5	1	8	45
Cabbage, raw	1 cup	1	tr	5	25
Carrot, raw	1 carrot	1	tr	5	20
Carrots, cooked, diced	1 cup	1	tr	10	45
Cauliflower, cooked	1 cup	3	tr	5	25
Celery, 8″ stalk	1 stalk	tr	tr	2	5
Corn, ear, 5″ x 1¾″	1 ear	3	1	16	70
Cucumber, pared	1 cucumber, 10 oz	1	tr	7	30
Lettuce, Iceburg, 4¾″ diameter	1 head	4	tr	13	60
Onions, raw	1 onion, 2½″ diameter	2	tr	10	40
Green peas, cooked	1 cup	9	1	19	115
Baked potatoes	1 potato, ⅓ lb	3	tr	9	90
Boiled potato	1 potato, ⅓ lb	3	tr	23	105
French fried potatoes 2″ x ½″ x ½″	10 pieces	2	7	20	155
Mashed potatoes, milk and butter added.	1 cup	4	8	24	185
Potato chips (medium) 2″ diameter	10 chips	1	8	10	115
Spinach, cooked	1 cup	5	1	6	40
Baked sweet potato, about 6 oz	1 potato	2	1	36	155
Tomatoes, raw about ⅓ lb	1 tomato	2	tr	7	35
Tomato juice, canned	1 cup	2	tr	10	45
Turnips, cooked, diced	1 cup	1	tr	8	35
Apples, about ⅓ lb	1 apple	tr	tr	18	70
Applesauce, canned, sweetened	1 cup	1	tr	60	230
Apricots, canned in heavy syrup	1 cup	2	tr	57	220
Bananas, raw about ⅓ lb	1 banana	1	tr	23	85
Blueberries, raw	1 cup	1	1	21	85
Cantaloupe, raw 5″ diameter	⅓ mellon	1	tr	14	60
Cranberry sauce, sweetened	1 cup	tr	1	104	405
Dates, fresh or dried	1 cup	4	1	134	490
Fruit cocktail, canned in heavy syrup	1 cup	1	1	50	195
Grapefruit, white	½ grapefruit	1	tr	14	55
Grapefruit juice, fresh	1 cup	1	tr	23	95
Grape juice	1 cup	1	tr	42	165
Lemonade, concentrated water added	1 cup	tr	tr	28	110
Oranges	1 orange	2	tr	16	60
Frozen orange juice concentrated, water added.	1 cup	2	tr	27	110
Peaches	1 peach	1	tr	10	89
Peaches, canned, in heavy syrup	1 cup	1	tr	52	200
Pears, raw	1 pear	1	1	25	100
Pears, canned, in heavy syrup	1 cup	1	1	50	195

TABLE 16–2. *Carbohydrate, Fat, Protein, and Calorie Contents of Selected Foods*—Con.

Item	Measure	Protein (gm)	Fat (Total Lipid) (gm)	Carbo-hydrate (gm)	Calories
Prunes, cooked, unsweetened	1 cup	2	1	78	295
Prune juice, canned	1 cup	1	tr	45	170
Raisins, dried	1 cup	4	tr	124	460
Strawberries, raw	1 cup	1	1	13	55
Watermelon	1 wedge 4" x 8"	2	1	27	115
Biscuits	1 biscuit	3	6	17	140
Cracked wheat bread	1 slice	2	1	12	60
White bread, enriched	1 slice	2	1	12	60
Chocolate cake, fudge icing	2" sector	5	20	67	445
Gingerbread, 2" x 2" x 2"	1 piece	2	6	29	175
Cookies, plain and assorted	1 cookie, 3" diameter	1	5	18	120
Cornflakes, plain	1 oz	2	tr	24	110
Corn muffins	1 muffin	3	5	23	150
Graham crackers	4 small or 2 medium	1	1	10	55
Doughnuts, cake-type	1 doughnut	1	6	13	125
Macaroni & cheese, baked	1 cup	18	24	44	470
Rolled oats	1 cup	5	2	23	130
Pie (1/7 of 9" diameter pie):					
Apple	1 sector	3	15	51	345
Custard	1 sector	8	14	30	280
Mince	1 sector	3	16	56	365
Rice-white, cooked	1 cup	3	tr	41	185
Spaghetti w/meat balls & tomato sauce.	1 cup	19	12	39	335
Shredded wheat	1 oz	3	1	23	100
Wheat germ	1 cup	18	7	32	245
Butter	1 pat or square	tr	6	tr	50
Lard	1 tbsp	0	14	0	125
Margarine	1 pat	tr	6	tr	50
Salad or cooking oils	1 tbsp	0	14	0	125
French dressing	1 tbsp.	tr	6	3	60
Mayonnaise	1 tbsp	tr	12	tr	110
Fudge, plain	1 oz	1	3	21	115
Hard candy	1 oz	0	tr	28	110
Jams, marmalade, preserves	1 tbsp	tr	tr	14	55
Granulated sugar	1 tbsp	0	0	12	45
Beer	1 cup	1	0	9	100
Ginger ale	1 cup	0	0	18	70
Cola-type beverage	1 cup	0	0	24	95
Gelatin dessert, ready-to-eat, plain	1 cup	4	0	34	140
Cream soup (mushroom)	1 cup	2	10	10	135
White sauce	1 cup	10	33	23	430

Adapted from Home and Garden Bulletin No. 72, U.S. Department of Agriculture, Sep. 1964.

TABLE 16–3. *Recommended Dietary Allowances of 17 Vitamins and Minerals*

Vitamins and Minerals	Units of measurement	Adult, or child 6 years of age or older
Vitamin A	U.S.P. units	5,000
Vitamin D	.....do	400
Vitamin E	International units	30
Ascorbic acid (vitamin C)	Milligrams	70
Thiamine (vitamin B_1)	.....do	1.2
Riboflavin (vitamin B_2)	.....do	1.7
Niacin or niacinamide	.....do	22
Vitamin B_6	.....do	2
Folic acid	.....do	0.05
Pantothenic acid	.....do	10
Vitamin B_{12}	Micrograms	5
Calcium	Milligrams	800
Phosphorus	.....do	800
Magnesium	.....do	300
Iron	.....do	15
Iodine	.....do	0.15
Copper	.....do	2

From Fact Sheet, Food and Drug Administration, U.S. Department of Health, Education, and Welfare, 17 June 1966.

EXERCISE AND RECREATION

The Flight Surgeon, more than any other individual, is in a position to help aircrewmen with fitness problems. He should (1) explain the values of exercise and recreation to airmen, (2) aid in the planning of fitness programs *which will be accepted,* (3) insure that facilities for recreation and exercise are adequate. The first function can be accomplished through lectures, movies, notices, and individual discussions.

The Flight Surgeon will be called upon to fulfill the second function either at the request of individuals or at the request of commanding officers. The Navy's position is clear: *voluntary* intramural and intermural sports programs are desirable but *compulsory* corrective action must be taken in cases of flagrant disregard for essential physical fitness. BUPERS Instruction 6100.2 series outlines a physical fitness program, standards, and a remedial program for naval personnel under 40 years of age. All airmen under 40 must meet the minimum standards set forth in this directive, or be compelled to participate in a remedial program. Individual preferences are certainly a consideration in any remedial program and these preferences, if compatible with the goal of achieving a minimum fitness, should be honored.

The most important consideration in program planning is to choose a program which will be fulfilled. For this reason intramural team sports and individual sports such as golf, tennis, bowling, cycling, rowing, swimming, and hiking are recommended. If an individual enjoys calisthenics, weight training, or running, these are excellent forms of exercise. If they also serve as a form of relaxation and recreation, and will be continued, they should be encouraged. Any program should be performed regularly three to five times a week. An occasional hard workout on the weekend is of little value to overall physical fitness. In fact, sporadic exercise probably does more harm than good.

Many aviators find that light exercise after a flight helps to relieve tensions. Because of the automatic action of the autonomic nervous system under stress, fliers frequently find that they are physiologically and mentally prepared for physical activity at a time when physical activity is impossible. Postflight exercise is an excellent way to return bodily functioning to normal. Very light exercise such as stretching or walking, if possible, or even some of the isometric exercises mentioned below, will serve the same purpose while flying—but not at altitude.

Unfortunately the persons who need exercise most are those who are least interested in it. Exercise plays an important role in weight control. It is as important to indoctrinate out-of-shape, overweight, uninterested individuals in the recreational values of physical fitness, the role of exercise in weight control, and the need for forming good fitness habits as it is to require a particular regime of exercise. A successful program cannot be accomplished without the proper attitude and cooperation of the individuals involved. The purpose of exercise in naval aviation is not to develop marathon runners or football stars, but to develop airmen who are physically fit to perform their duties. Some fliers erroneously believe that exercise is less important as they get older. Many of the degenerative effects so often unjustly attributed to passing time per se can be partially offset through a carefully planned exercise program.

One particular type of exercise is worth mentioning here. A great deal of publicity has presented isometric contractions as the lazy man's answer to fitness. Isometric contraction—the pitting of a muscle or set of muscles against an immovable object or another set of muscles for a brief period—has been advocated as a means of keeping in shape without doing anything.

There is some evidence that much of the muscle growth occurs in the first six to eight seconds of muscle exertion. Isometrics, although not so miraculous as some have often claimed, can serve as a useful supplement to any physical fitness program.

A Department of the Navy pamphlet (*Shape Up,* 1962) presents a series of nine isometric exercises which are meant to be used in conjunction with the Navy self-conditioning program of five exercises (the stretcher, situps, pushups, sustained jumping, and stationary run). This program will meet the needs of many Navy personnel.

The following are the nine recommended isometric exercises. Each exercise should be performed for about six seconds, and the breath held during this period. It is advisable to relax completely for a few seconds before going on to the next exercise. For the first week, one should apply only about 50 percent maximum effort in these push or pull positions, maintaining tension for approximately six seconds only in each position. For maximum benefit, these exercises must be performed every day.

1. *The Pull-Up* (for arms and shoulders). Sit straight, grasp the sides of the chair tightly with both hands and pull up as hard as possible.

2. *The Hand Press* (for arms, chest, and shoulders). Sit straight with chest out and arms held across chest, place one fist inside the other. Press together using all the strength of the arms and the shoulders.

3. *The Back Pull* (for the back). Keep back straight and lean forward until you can grasp your legs on braces of chair. Pull straight up using back muscles only.

4. *The Neck Presser* (for the neck). Sitting straight, clasp the hands behind the neck holding elbows forward. Pull forward with the hands and at the same time press the head backwards.

5. *Tummy Tightener* (for waist and abdomen). Sitting with legs together straight out, bend forward and grasp the legs just below the knees. Press down with the hands, at the same time press up against the hands with both legs.

6. *The Criss-Cross* (for chest and legs). Placing the feet about 4 inches apart, bend forward and place hands against inside of opposite knees. Attempt to press knees together while at the same time, holding them apart with the hands.

7. *The Body Lift* (for shoulders, arms, abdomen). Keeping the back straight, lean forward and place the hands palms down against the side of the

chair. Hold legs straight out, attempt to raise body about one inch off the chair.

8. *The Leg Squeezer.* While sitting forward on the edge of a chair, lean back, hold legs straight out. Hook one foot over the other and hold tightly. Rest feet on floor, keep legs straight, then try to pull the feet apart.

9. *The Arm Curl* (for the upper arms). Sit straight, grasp the underside of a heavy desk or table with palms up, forearms parallel to desk. Push up as hard as possible.

The third function of the Flight Surgeon with regard to exercise and recreation is to insure that exercise and recreation facilities are adequate. Obviously, the Navy has unique problems in this area. The extensive participation in volleyball in the Navy, for example, is the result of carrier space limitations which preclude other sports such as softball. In many cases, an individual or group exercise-recreation program must be adapted to meet environmental requirements. The environment may possibly limit, but should not prevent, physical activity. Fortunately, the Navy, because of its commitment to fitness and recreation through exercise, has provided excellent facilities at most shore stations.

SLEEP AND REST

Neuromuscular fatigue is alleviated and mental alertness restored by sleep in favorable surroundings. Noise, excitement, and worry adversely affect the restfulness and diminish the benefits of sleep. While the optimum duration of sleep varies widely throughout life, the normal requirement for flying personnel is approximately eight hours in every twenty-four-hour period. During any period of heavy or prolonged scheduling of aviators, the Flight Surgeon can be of great value to the Operations Officer. By consulting personally with him, the Flight Surgeon can help by estimating when grounded aviators will be returned to duty, and in many other ways. If—in the Flight Surgeon's opinion—certain aviators are being overscheduled to the point that adverse effects may be expected, he should discuss this problem with the Operations Officer.

In unusual flight situations, the Flight Surgeon should provide guidance with regard to provisions for sleep and rest (OPNAV Instruction of the 3740.7 series).

FATIGUE

One of the causes of faulty flier performance at the end of a flight is fatigue (Bartlett, 1942; Davis, 1948; Drew, 1940). Although everyone has some idea of what is meant by the term "fatigue," and most have experienced something which they have called "fatigue," a satisfactory and precise definition of fatigue has not been formulated. Because of the diverse nature of fatigue, most studies in the area suffer from narrowness of scope and are only moderately applicable to aviation. In order to circumvent these problems, the term "operational fatigue" has been coined to describe the combined detrimental effects of muscular exertion, anxiety, and boredom on the performance of airmen. This broad concept of fatigue, although unscientific, is meaningful in the context of aviation.

The Physiological and Psychological Bases of Fatigue

Any activity, including sleep, results in an oxygen cost, an expenditure of energy, and a heat output. The "cost" to the body of oxygen used, energy expended, and heat produced, as they relate to activity, is presented in table 16–4. As work increases, the body becomes more fatigued. Mental activity and emotions also increase fatigue. Ultimately, true relief can only come in the form of sleep, although some drugs will temporarily allay fatigue effects.

TABLE 16–4. *Classification of Physical Activity by Its Severity (average values for 69″, 167 lb. man)*

Activity	Oxygen Cost lb O_2/hr	Energy Expenditure Kcal/min	Heat Output Btu/hr
Sleep	0.05	1.2	280
Very Light Work	0.05–0.10	1.2–2.5	280–595
Light Work	0.10–0.19	2.5–5.0	595–1190
Moderate Work	0.19–0.28	5.0–7.5	1190–1785
Heavy Work	0.28–0.38	7.5–10.0	1785–2380
Very Heavy Work	0.38–0.47	10.0–12.5	2380–2975
Unduly Heavy Work	over 0.47	over 12.5	over 2975

Adapted from Fletcher, 1964.

There are certain demonstrable physiological changes which occur with fatigue. These changes, due to muscular activity, mental activity, and emotions, are, in a sense, the "bases" for fatigue. Airmen perform very little physical work. According to the classification used by Fletcher (1964), most flying activity is considered light work. Lactic acid does not accumulate to any extent during a flight, and the glucose level is lowered only slightly (McFarland, 1953). It seems unlikely that muscular exercise as described by Chapman and Mitchell (1965), Karpovich (1959), and Morehouse and Miller (1959) contributes much to the fatigue experienced by airmen. Mental activity also uses very little energy. McFarland (1953) cites a study by Benedict and Benedict which demonstrated that mental activity for several hours uses only the amount of calories found in half a peanut. But the fact remains that airmen show fatigue symptoms during and after flights.

Operational Factors Contributing to Fatigue

Almost all stressors associated with aviation contribute to fatigue, either through muscular or mental activity, or through the relevant psychological and physiological mechanisms. These stressors are well known. Vibration, acceleration, heat, and oxygen shortage, for example, are a few of the factors which contribute to muscular fatigue. Of more immediate concern, however, are the problems of mission scheduling. These problems fall into two related classes: duty-rest ratios and the upset of diurnal cycles. When possible, duty-rest ratios should correspond to the eight hours on—sixteen hours off to which most individuals are accustomed. When operational considerations make deviations from this pattern necessary, special preparations for the airmen should be made. Airmen should be regularly monitored for the effects of fatigue and, if necessary, grounded.

Marchbanks (1960) studied flying stress during four 20-hour flights. He found that missions which commenced in late evening required more adjustment and resulted in more stress than missions scheduled during the day. This is one type of upset to the diurnal cycle which should be avoided when possible. Another type is what Juin (1962) calls "timetable readjustment." Jet transport aircraft have made it necessary to upset diurnal, eating, and digestive cycles considerably by flying so far so rapidly. Layovers which require sleeping during the day in strange quarters and in a different climate or eating at odd hours may adversely affect tolerance to fatigue. The Flight Surgeon should do everything possible to insure that acceptable quarters and meals are available during extended cross-country flights. Provisions for inflight meals and sleeping should be made whenever possible.

Identification of Excessive Fatigue

Fatigue results in reduced physiological, physical, and mental capacity. These reduced capabilities will be manifested in a deterioration in any or all aspects of flying performance. For this reason the Flight Surgeon must be conscious of the symptoms, often insidious, indicative of fatigue. The onset of fatigue may be gradual or rapid, immediate or delayed. Fatigue may be temporary (acute) or prolonged (chronic). Obviously, chronic fatigue makes an individual more susceptible to acute fatigue. It is well known that objective measures of fatigue and subjective reports of fatigue rarely coincide. The Flight Surgeon should be able to recognize fatigue by means other than an aviator's subjective report.

Bartlett (1942) found that as fatigue increased, performance standards deteriorated, yet pilots showed increased euphoria. Although pilots accepted poor performance, they did not realize that their performance was suffering. Timing at first improved, but then deteriorated. Gross errors appeared, continuity of performance was lost, and peripheral information was ignored. In addition, fatigue may result in general irritability, gastric disturbances, insomnia, deterioration in reasoning and judgment, sensory and motor hyperactivity, shortness of breath, and lowered morale. Any or all of these may be useful indicants of fatigue.

Other crewmembers, associates, and the family of the airman may be helpful to the Flight Surgeon in diagnosing fatigue before it reaches a level which could lead to an accident. The identification and control of fatigue is an important, but often neglected aspect of accident prevention.

Control of Fatigue

Control of fatigue is a complex problem requiring the cooperation of airmen, their wives and families, the Flight Surgeon, and the Commanding Officer. An appropriate regimen to minimize fatigue effects must attend to many factors of diet, exercise, recreation, sleep, and operational scheduling.

DONATION OF BLOOD

Since aircrew personnel are exposed at all times in flight to unusual demands for oxygen-carrying blood, certain restrictions should be placed on persons who have donated blood. OPNAV Instruction 3740.7 series entitled "Fitness of Flying Personnel" provides guidance regarding the period of grounding and flight limitations. As a rule, flying personnel should be grounded for four days following donation of 500 cc of blood. Blood should not be donated within four weeks of anticipated combat operations or operational flights from aircraft carriers. This is to preclude possible lowered resistance to fatigue or to injury or to illness. Therefore, the carrier and combat aviator should restrain his altruistic impulses when asked to donate blood, except in an emergency.

MEDICATIONS

It is the responsibility of the Flight Surgeon to monitor the use by flying personnel of drugs and other substances which might be detrimental to their flying ability. Of considerable concern is the problem of self-medication. Many commonly used medications, available without prescription, produce side effects which are considered dangerous for flying. Flight Surgeons must maintain a vigorous information and orientation program designed to demonstrate the dangers of self-medication.

Of value to the Flight Surgeon in specifying the period of grounding necessary following drug use is a publication by the (then) Federal Aviation Administration (1962) entitled "Guide to Drug Hazards in Aviation Medicine." This guide lists approximately 2,000 drug agents, briefly describes the undesirable effects of each drug to aviation personnel, and specifies the period during which airman duties are contraindicated.

The administration of any drug must be monitored by the Flight Surgeon in terms of its likely effect on flight personnel. There are certain classes of drugs, however, which are of particular concern, in many instances because of their easy availability and the consequent likelihood of being used without medical direction. This section describes the more important classes.

Antihistamines and Decongestants

The use of self-medications containing antihistaminics is especially dangerous for flying personnel. Response to such drugs can vary from no effect at all to drowsiness, reduced alertness, and even mental depression. These drugs also may affect depth perception, the operation of the vestibular apparatus, and the proficiency of perceptual-motor skills.

Closely allied, in terms of use, to the antihistamines are nasal decongestants. Indiscriminate use of nasal decongestants may produce tachycardia and nervous states. Mydriasis also may be produced, representing a possible decrease in visual capability. Naphazoline hydrochloride appears to be the most dangerous of the nasal decongestants.

Antibiotics

Under most circumstances, use of antibiotics can be controlled by the Flight Surgeon. On occasion, however, an airman may receive these drugs from a civilian physician or may use some left over from a previous illness. Streptomycin, dehydrostreptomycin, and Chloromycetin require special consideration in aviation medicine. The first of these may produce vertigo, deafness, and other symptoms of eighth nerve damage. Dehydrostreptomycin, although less toxic than streptomycin, does not give a warning of dizziness. Chloromycetin can damage the erythropoietic mechanism which adversely affects the oxygen-carrying system. Sulfa drugs also can affect the bone marrow and the oxygen-carrying capacity of blood.

Tranquilizers

Tranquilizers may be quite dangerous when used in the aviation environment, since a certain amount of anxiety and awareness has a definite physiological benefit in a stressful situation. A pilot should be able to appreciate, both rationally and emotionally, the risks of a particular course of action.

Psychological testing indicates that use of tranquilizers may produce highly significant alterations of judgment and orientation to reality. Airmen should be removed from flying status while using tranquilizers. It also may be advisable to continue the period of grounding for some time following the cessation of use.

Antimalarial Drugs

In certain zones of operation, it may be necessary to use antimalarial drugs such as chloroquine and primaquine for suppressive and therapeutic purposes. There is no evidence that these drugs have sufficient toxicity in recommended dosages to contraindicate their being administered to flying personnel under appropriate conditions (Haas, 1955). However, inasmuch as there are reports that antimalarial drugs may cause visual difficulties, the Flight Surgeon would be wise to monitor personnel closely while the drug is being used.

Motion Sickness Drugs

Inasmuch as all motion sickness drugs appear to have serious side effects which would affect flying performance, their use for flight personnel is not advisable. The only exception might be for student aviators during the initial stages of training when they fly only with instructor personnel. Even then, use of these drugs should be monitored closely by a Flight Surgeon.

Meclizine (Bonine) and cyclizine (Marezine) are the most effective of the antihistamine motion sickness drugs, and their side effects are milder. There are, however, substantial individual differences in response to these drugs, thereby making the choice of a particular drug for a particular individual rather difficult.

STIMULANTS

Aviation activities demand total alertness of aircrewmen, even though missions may last for many hours. Inasmuch as agents exist which will postpone to some extent the onset of fatigue, a Flight Surgeon must be prepared to make decisions concerning the advisability of using such agents.

Central Nervous System Stimulants

Central nervous system stimulants, such as amphetamine, dextroamphetamine, and methamphetamine, will postpone fatigue and allow persons to remain alert for many hours longer than would be the normal case. In well-integrated subjects, these drugs do not appreciably affect physical or mental efficiency, but do allow work to continue over longer than normal periods. Ordinarily, the dose is 2.5 mg of dexedrine sulfate repeated not more frequently than every three hours, or 5 mg repeated not more frequently than every six hours (Department of the Air Force, 1962).

The use of amphetamines to increase or prolong work output must be closely supervised by the Flight Surgeon. Strickland (1961) refers to this type of drug as indeed a "two-edged sword." A Flight Surgeon must balance the possible effects of performance impairment due to fatigue against the increased energy expenditure as the body is forced to perform at peak proficiency for periods longer than normal.

Amphetamines also may be prescribed as anoretic agents in weight control programs. With aviators, it is important that the schedule for use of this drug is appropriate for the schedule of flight activities. In general, an aviator should be grounded while taking anoretics for weight control.

Amphetamine drugs are not habit forming in the physiological sense. However, individuals can develop a dependence on such drugs, which in itself would represent a dangerous situation. Overdosage of these drugs will produce excessive excitement, headache, and sleeplessness, and will occasionally interfere with recall.

Coffee

The principal pharmacologically active ingredient in coffee is a methylated xanthine derivative caffeine. Caffeine affects voluntary and involuntary muscles, the kidneys, and other organs, but its main effect concerns the response of the central nervous system and heart muscle to this drug. In moderate doses, caffeine acts to increase performance levels and to lessen the effects of fatigue (Huene, 1964).

A cup of coffee contains approximately 100 to 150 mg of caffeine, an amount representing an average U.S.P. dose. Studies have shown a small but significant correlation between certain heart disorders and the use of coffee, when such use exceeds roughly five cups per day. This is understandable since it is known that excessive doses produce cardiac arrhythmias. Also, *excessive* coffee drinking causes gastric hyperacidity and is frequently associated with chronic gastrointestinal complaints. Flight Surgeons, therefore, should stress moderation in the use of coffee, as in virtually everything.

SMOKING

The principal problem in aerospace medicine produced by smoking concerns the extent of the loss of night vision. As discussed in the section on toxicology, the carbon monoxide from cigarettes combines with hemoglobin and reduces the oxygen-carrying power of the blood. Thus, smoking increases any deleterious effects of hypoxia and, due to the poor vascularization of retinal areas, reduces night vision capability.

Evidence now exists that smoking has a more general effect on personal well-being Lung cancer is the most dramatic of the effects attributed to smoking, but it also has been cited as a cause of cancer of the lip, mouth, and larynx.

Cigarette smoke causes irritation to the epithelial lining of the respiratory tract, and thus edema and swelling, preventing air from passing in and out freely.

Cigarette smoking also affects the cardiovascular system, because of its potent vasoconstrictive effect. The smoke of one standard-size cigarette will cause the skin temperature in the foot to be lowered by about five degrees F. In addition, there is evidence that nicotine in the bloodstream will cause the blood to clot faster and more easily.

Flight Surgeons should see that all aviators are informed concerning these effects of cigarette smoking.

ALCOHOL

Ingestion of specified amounts of alcohol results in severe toxic reactions in man and seriously degrades his performance capability in flying an aircraft. The problem has two aspects in terms of hazard to safety: (1) flying while under the influence of alcohol and (2) flying while still "hung over" from the aftereffects of alcohol. In some respects the later is the more serious problem, since overt symptoms of intoxication are not readily observable, nor does the individual appear to be much different from his usual self.

The ingredient in spirits that accounts for most of the physiological effects is ethyl alcohol. There are also lesser amounts of other volatile substances (congeners), chiefly, higher alcohols, aldehydes, acids, ketones, and unknown substances. While not toxic in the amounts present, they serve to slow down the rate at which alcohol is disposed of by the body. Alcohol is fast acting, absorbed into the bloodstream without requiring digestion, and appears in the blood only minutes after ingestion. The concentration of alcohol in the blood rises rapidly during the initial period of absorption, peaking at about one-half to two hours later, then slowly declining as the rate of elimination exceeds the rate of absorption. A feature of significance for aviation is that a considerable percentage of the ingested alcohol remains in the blood for lengthy time periods following drinking. McFarland (1953) cites research indicating a significant quantity of alcohol in the blood 16 hours after drinking.

The concentration of alcohol and rate of its absorption are influenced by various factors, foremost of which are: the total amount of alcohol intake; the quantity and kind of food in the stomach prior to alcohol intake; the total time period in which the alcohol is ingested; and the kinds of liquor ingested. To some extent, individual differences govern the speed with which alcohol is absorbed and the symptoms and effects of intoxication.

Various levels of blood alcohol are used to describe degree of intoxication, hence, impairment in behavior.

below 0.05%	no impairment
0.05—0.15%	possible influence on behavior
above 0.20%	significant influence on behavior
above 0.30%	dazed, disoriented condition
above 0.40%	insensible
0.50%	death

Effects of Alcohol on Man

Alcohol is a depressant rather than a stimulant, affecting the central nervous system most prominently, although other parts of the body are involved. With alcohol intake, pulse rate, blood pressure, and depth of breathing may increase but gradually return to normal. The small blood vessels in the skin become dilated, resulting in increased blood flow to the skin, hence the feeling of warmth and stimulation. Respiration is not affected unless large amounts are consumed. Alcohol depresses cell oxidation in that the tissue cells cannot use oxygen efficiently. These histotoxic effects are similar to those of other narcotics which inhibit the oxidation of substances important in carbohydrate metabolism in brain tissue. Thus, at altitude, the effects of alcohol are more pronounced than at sea level. Oxygen want in conjunction with alcohol in the system has serious effects on nervous tissue and consequently on behavior. This was demonstrated by McFarland and Forbes (1936) in a study of the combined effects of alcohol and oxygen lack at high mountain altitudes. Figure 16–1 shows the effect of altitude on the metabolism of alcohol. The disadvantage of alcoholic intoxication is that for any given altitude, the physiological altitude is much higher when there is alcohol in the blood.

Alcohol adversely affects sensory, motor, and intellectual functions. McFarland (1953) has outlined various patterns of behavior that emerge with

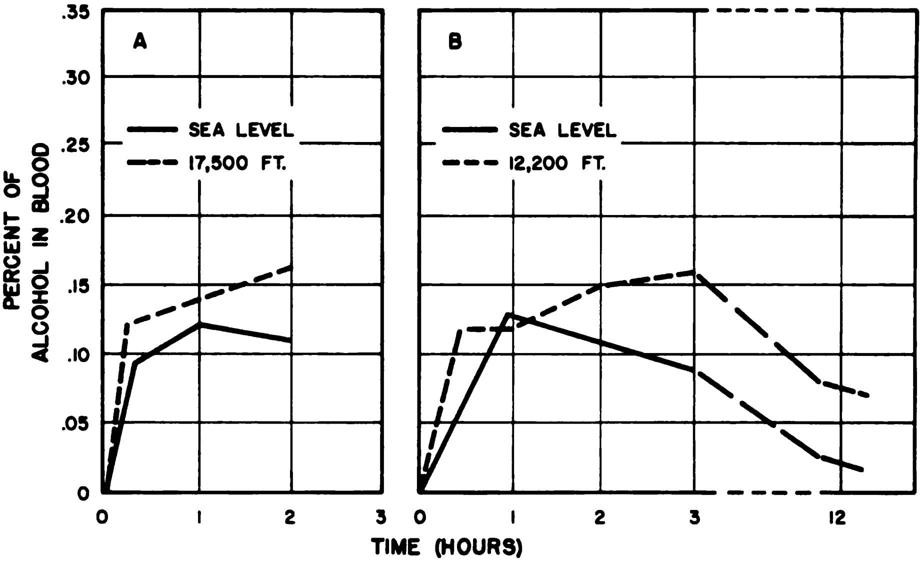

FIGURE 16–1. *The effect of altitude on the metabolism of alcohol.* (REPRINTED FROM "THE METABOLISM OF ALCOHOL IN MAN AT HIGH ALTITUDES," BY R. A. MCFARLAND & W. H. FORBES, PAGES 387–398 IN Human Biology, VOL. 8, 1936, BY PERMISSION OF THE WAYNE STATE UNIVERSITY PRESS.)

successive stages of intoxication. The initial symptoms are a dulling in critical judgment and sense of responsibility. Inhibitions are released, and the intellectual functions are impaired. With increases in intake, degradations occur in skilled responses, sensory perception, and motor control. Finally, insight is lost concerning the extent of impairment.

Impairment in motor functioning (including visual responses) is due to the effect of alcohol on nervous control. Error frequency increases considerably while reaction time is slowed. The sensory function of vision is most impaired by alcohol. The visual fields are constricted and the ability to see at low levels of illumination is reduced. Alcohol also adversely affects intellectual functions such as memory, judgment, and reasoning.

If one could describe a classic picture of performance decrement that spells disaster for the pilot, it is the syndrome of alcohol intoxication.

Alcohol Hangover

A serious hazard to safety results from flying while suffering from a hangover caused by the aftereffects of alcohol. The problem is insidious in that the individual is definitely under the influence of alcohol although most of the overt signs of intoxication are not readily observable. Performance impairment is still considerable during this period and the possibilities of errors in flight are tremendous.

Since considerable quantities of alcohol remain in the blood for as long as 16 hours following a drinking session, the need for an established time period between drinking and flying is obvious. Present-day convention stipulates that when more than four ounces of alcohol are ingested, at least 18 hours, and preferably 24 hours, should elapse before flying. This does not apply to drinking in moderation: for example, two ounces or less of alcohol have no impairing effect on performance.

Realistically speaking, the time period between drinking and resuming flying is difficult to monitor and enforce; thus, it behooves the Flight Surgeon to be aware of the drinking habits of the flying personnel under his control. Also, a discussion of the subject, alcohol and flying, should be a part of his program for the indoctrination and training of aircrew. The seriousness of the impairment resulting from alcoholic intoxication necessitates this.

Elimination of Alcohol

The human body requires about three hours to eliminate one ounce of alcohol. This rate of elimination is fairly constant and is not hastened by use of any drugs, by any procedures regarding eating of meals before or after, or by any amount of sleep or coffee. Coffee, however, as a result of its capability for central nervous system stimulation, does tend to counteract the depressant effect. It is important that aviators understand that, although the effects of alcohol on a full stomach are decidedly less than on an empty stomach, the rate of elimination is identical in either case. Since different drinks contain varying amounts of alcohol, the time required to recover depends both on the number and kind of drinks. Figure 16–2 shows the approximate number of hours required for the elimination of the alcohol in a number of drinks of different kinds.

Treatment of Alcohol Problems

Most problems with alcohol are best treated, or avoided, by providing proper information to aviators concerning the action of alcohol. Ingraham

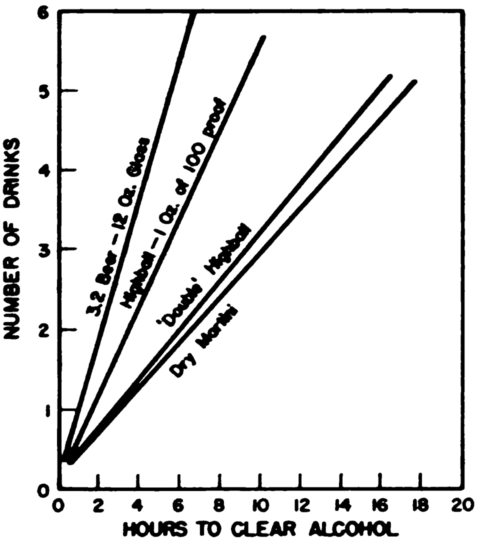

FIGURE 16–2. *Approximate time required for elimination of alcohol from various kinds of drinks* (INGRAHAM, 1965).

(1965) suggests the important points to remember when dealing with alcohol and flying are:

1. An amount of alcohol which produces no effect at sea level can cause disastrous impairment of judgment and coordination at 10,000 feet.

2. There should be absolutely no alcohol in the blood when a person flies. He must abide by the one ounce every three hours rule.

3. The rate of elimination of alcohol is fixed and cannot be influenced by any specific actions of the person involved. Three hours are required to clear one ounce of alcohol.

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CHAPTER 17

Aerospace Toxicology

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INTRODUCTION

THE PRACTICE of aerospace medicine includes a concern for those chemicals used in naval aerospace operations which are toxic or which give rise to toxic products under conditions of flight or occupational use.

In order to evaluate toxicological problems effectively, a Flight Surgeon must be conversant with the principles and practices of industrial medicine, since aviation toxicology embraces both conventional toxicological problems and unique problems associated with flight. There is no sharp distinction between the two as to the toxic substances involved. Occupational toxicology is concerned with hazards and diseases resulting from a broad spectrum of toxic agents, and much data is available (see Fleming, D'Alonzo, & Zapp, 1960; Patty, 1958, 1963; Gafafer, 1964).

Aerospace operations are characterized by rapid increases in technology. As new materials and chemicals are produced, they may be introduced into use because of desirable characteristics without full knowledge of possible effects. The Flight Surgeon must remain alert for instances requiring information not yet available in the archives of industrial medicine.

The Flight Surgeon also has a requirement to train aircrew personnel in toxicology. He must provide them with knowledge of potentially harmful materials with which they may come in contact, principles and safeguards in exposure control, and must develop in them an awareness of the possible hazards so that they may institute emergency measures when needed.

PROBLEMS IN AEROSPACE TOXICOLOGY

Important Considerations

There are several important aspects of aerospace toxicology which must be considered in establishing appropriate prevention and treatment programs. These are listed briefly below and discussed in greater detail in later sections as they relate to specific toxic agents.

1. *Acute versus Chronic Toxicity.* The most important toxicological incidents in aerospace operations are acute exposures occurring during flight, characterized by short-term, high-level exposure to toxic agents. Changes in the chemical composition of the cabin air may occur subtly and/or quickly due to changes in altitude and ventilation rates, or due to the generation of toxic substances resulting from leakage of fluids or from fire.

Acute inflight exposures can be considered as two types: First, there is the exposure characterized by a sudden incapacitation of the individual. Second, there is the exposure which results only in small changes in performance capability. There is evidence which suggests, if only indirectly, that chemical agents have contributed to accidents classified as caused by pilot error. The pilot's job is a demanding one. In some segments of flight, the balance between critical tasks and human abilities is delicate even for well-trained crews. Thus, any performance impairment due to noxious gases becomes a matter of considerable concern.

In the ground support situation, the concern is for chronic exposure to potentially toxic agents during day-to-day work. There also are special problems associated with the handling of various substances aboard aircraft carriers due to continuous storage of chemicals in confined spaces and in proximity to working personnel. The flightline operation in aviation is similar to the industrial environment with the exception that a lesser number of toxic agents are involved.

2. *Time/Dose Relationship.* With most substances, the medical severity of an exposure is a function of a reciprocal relationship between the duration of exposure and the concentration of the chemical. As concentration increases, there is a shorter period of allowable exposure time prior to the onset of symptoms.

Qualitative changes in the effect of certain chemicals may occur as concentration increases. For example, at high concentrations various acid gases are so irritating to the upper respiratory tract that often the individual escapes from the environment before damage to the alveoli or any absorption occurs. Yet, some gases (e.g., nitrogen dioxide from rocket fuel) do not irritate the upper respiratory tract and yet will produce pulmonary edema.

3. *Physiochemical Factors.* Substances which enter the bloodstream tend to be selectively absorbed in certain organs or tissues. For example, fat-soluble compounds tend to accumulate in the nervous system. Heavy

metals, on the other hand, tend to produce damage at the portal of exit, resulting in kidney damage.

4. *Portal of Entry.* The two most common portals of entry in aerospace medicine are the respiratory tract and the skin. The gastrointestinal tract is of lesser concern. In acute exposures, the toxic agent enters mainly through the respiratory tract. In chronic exposures, the skin frequently is the important portal of entry.

5. *Preexisting Conditions.* Susceptibility to the action of a toxic agent may be greatly increased in the event of any organ impairment. Disease or damage to structures such as the liver or the lungs presents an obvious example. In addition, the toxic effect of a given concentration of a chemical can increase if additional toxic agents are present. Thus, a low concentration of carbon monoxide is more damaging if another agent is interfering with the gas-transport capabilities of the blood.

6. *Individual Variability.* As is clearly shown in allergic reactions, there is considerable variability in the responses of indivduals to specific toxic agents. Thus, in a given working environment in which a number of persons are in relatively constant contact with a low-concentration chemical, only one may exhibit symptoms indicating a toxicological episode.

Allowable Degree of Bodily Impairment

It is desirable to be able to specify concentrations of toxic agents that aircrewmen could be exposed to without dangerous impairment of performance. Unfortunately, present methodology is inexact in defining the degree of bodily impairment which can be safely allowed in flight operations. Theoretically, even a slight degree of impairment is hazardous to a pilot's task. Even so, it is desirable to have some insight concerning the likely hazard when dealing with various chemical agents. For this reason, industrial medicine personnel have worked for many years to set standards for toxic substances which define the levels of concentration injurious to health. The following two designation systems have been developed:

Threshold Limit Values (TLVs) attempt to specify that a chemical substance of a given concentration (usually in parts per million for gases and vapors, and milligrams per cubic meter for fumes and dust) will produce no demonstrable effects on a man exposed to it eight hours daily on the job. The TLV is a time-weighted average concentration which will not adversely affect a man under conditions of repeated day-to-day exposure. These values are set periodically by the American Conference of Governmental Hygienists (Zapp, 1960) and cover a great number of substances, most of which are not important to the aviation environment. For a discussion of TLV, see Fleming et al. (1960) and Brewer (1965). Current TLVs are published in BUMED Instruction 6270.3.

Maximum Acceptable Concentrations (*MACs*) define limiting concentrations which should never be exceeded. These values, set by the American Standards Association, are related to but not identical to TLVs. MAC is an older designation, less useful than the TLV, and is not used in the Navy. Most MACs are based on chronic long-term effects, but some are based on short-term acute effects. Thus, quantitative comparisons of toxicity among agents are subject to error.

Literal application of TLVs or MACs is not recommended. For one thing, very few values have been established on the basis of examining humans under relevant environmental conditions. Most often, speculation and animal data are used in setting values. Other difficulties are that: toxic concentrations rarely remain constant for any given time period, individuals vary significantly in their susceptibility to toxic substances, and hazardous environments frequently contain mixtures of substances rather than single compounds. An important feature for the aviation environment is that the published values assume sea level conditions and do not consider the effects of altitude on toxicity. An additional difficulty results from making predictions based on a value without considering the time of exposure. For example, the TLV of carbon monoxide is 50 parts per million (ppm) but much higher exposure levels are permissible for short time periods. BUMED (BUMED letter, subject: Short-Term Threshold Limits for Carbon Monoxide in Naval Surface Ships, 15 October 1964) recommends the following emergency exposure levels: 1,500 ppm for 15 minutes; 800 ppm for 30 minutes. When the tasks to be performed require mental acuity and alertness, the levels are reduced to 1,000 ppm and 500 ppm, respectively. In view of the above constraints, the Flight Surgeon should consider the TLVs or MACs to be, at best, useful guidelines to follow on toxicological matters in aviation.

Finally, it is important to recognize that the maximal toxic potential of a substance is hardly ever realized in the individual. The toxicity observed is only a fraction of the substance's true toxicity. Immediately upon entry of a toxic substance, the body begins to reduce the concentration by processes of elimination. These include metabolic degradation, detoxification, and excretion. While there are certain exceptions to this (Stokinger, 1963), the Flight Surgeon must be familiar with the systemic courses of the well-known poisons and the manner in which the body attempts to correct the situation.

Criteria for Diagnosis

As mentioned earlier, the clinical symptoms of exposure to toxic substances are at times nonspecific to a given agent, which makes differential diagnosis a decidedly difficult task. This is particularly the case when small amounts of toxic agents are involved. Also, some chemical intoxications present symptoms which are similar to those produced by biological agents or to conditions of a degenerative nature. Thus, proper diagnosis may at times be difficult to achieve. As an aid to effective diagnosis, the following principles are useful:

1. Obtain a work history of the afflicted individual and any unusual circumstances in the use of suspected chemicals or materials. Obtain samples of the material.
2. Solicit a detailed expression of the individual's symptoms while looking for clinical symptomatology.
3. Obtain samples of human matter, breath analysis, etc. Take advantage of consultation and test facilities available.

Following such systematic procedures will facilitate differential diagnosis and subsequent treatment.

As an aid to diagnosis, Boysen (1961) has prepared a series of criteria to follow. These are:

1. The causative agent must be present in the environment in sufficient quantity to produce the symptoms. The quantity of the toxicant must be such that the amount absorbed will exceed the body's ability to excrete it.
2. The time-dose relationship must be adequate. With absorption, detoxification, and excretion occurring simultaneously, there must be a significant quantity present in the body to produce the symptoms.
3. The agent must be capable of producing the syndrome. The difficulty here is that small amounts of an agent may produce nonspecific symptoms, hence may be confused with other agents or with conditions unrelated to the environment.
4. Time between exposure and appearance of symptoms must be adequate. In most cases, the greater the amount of the material absorbed in a given time,

the shorter the latent period before the onset of symptoms. In general, the sequence of events should be known for each specific chemical.

Other causes for the condition must be ruled out. Every effort must be made to eliminate normal degenerative processes, infectious diseases, and other chemical toxicants.

PREVENTIVE MEDICAL MEASURES

Toxic substances most often result in less harmful effects than actual diseases. These effects, however, may seriously alter the performance of aircrews. Thus, the Flight Surgeon must be concerned with subclinical disease patterns. He must be constantly alert to the possibilities of human impairment resulting from exposure to toxic substances. The detection of subclinical disease is difficult at best. In order to facilitate detection and diagnosis, certain practices are recommended.

1. The Flight Surgeon must know the poisonous materials and the relative toxicity of various chemical compounds existing in the working environments. Information on the nature of these compounds, physical state, chemical properties, and other characteristics should be actively solicited from various expert individuals and sources. Another essential is knowing how toxic substances are absorbed and the routes of absorption.
2. Periodic physical examinations of flight personnel should be conducted and the results examined for trends. Enclosure 1 of Navy Civilian Personnel Instruction 792 specifies examination intervals for people working in hazardous environments. Analyses are also required of certain body fluids to determine levels of absorption and any successive incremental changes. Since the investigations deal with microquantities, the Flight Surgeon must coordinate with well-equipped clinical laboratories for analysis of materials.
3. The Flight Surgeon should know which personnel are subjected to the greatest hazard and also the work situation in which it occurs. This permits a concentration on selected individuals showing the greatest hazard exposure. This practice will aid in keeping examinations and analyses to a reasonable workload level while not compromising the control effectiveness.
4. Periodic inspections of the work environment must be made by the Flight Surgeon. These should be scheduled so as to see the actual work

being performed. It may be found that, unknown to supervisors, certain substitutions have been made in the chemicals being used. It may also be found that important safety procedures are not being followed.

Principles of Immediate Treatment

The prevention and avoidance of poisoning in the aviation environment are, in large part, aided by (1) the use of protective equipment and clothing and respiratory devices, (2) effective design of equipment in the use, handling, and storage of toxic materials, and (3) adequate ventilation and oxygen replacement. In the event of individual poisoning, prompt handling of the emergency will have a significant bearing on the outcomes of the exposure. The following immediate treatment procedures are recommended:

Skin Contact

Flush the skin with large amounts of water, lukewarm if possible.

Remove contaminated clothing.

Chemical burns of the eye should be flushed with large amounts of water for at least 15 minutes. The more rapid the flushing, the less severe will be the injury. A weak solution of one teaspoon of bicarbonate of soda to one quart of warm water is also recommended.

Inhalation

Remove the victim from the contaminated area.

Keep the victim quiet and warm, preferably lying flat.

Provide artificial respiration when indicated.

Administer oxygen if available.

Keep the victim's breathing passages open.

Pulmonary Edema

Administer oxygen by inhalator or nasal catheters. This is urgent and must be given in a concentration sufficient to keep the victim's color pink. (Keep him from becoming cyanosed.)

Venesection may be useful if cyanosis and venous engorgement are present. It is harmful if the victim becomes pale and goes into circulatory collapse.

Do not use drugs that depress the respiratory center or cardiac stimulants or drugs for increasing blood pressure.

Do not use sulfa drugs or antibiotics until the victim is free from signs of circulatory collapse or anoxia, and then only if pneumonia complications are present.

(Special first aid treatment and medical care for classes of chemicals can be found in various sources such as Fleming et al., 1960, and Johnstone & Miller, 1960.)

SUPPORT FACILITIES FOR TEST AND CONSULTATION

Various facilities are available to the Flight Surgeon for test and consultation on matters of occupational medicine and industrial hygiene. These services are available in accordance with the provisions of Navy Civilian Personnel Instruction 792 and the *Manual of the Medical Department* (part 26–10). Support is provided by the following installations:

Naval Preventive Medicine Unit 2, Norfolk, Va.
Naval Preventive Medicine Unit 5, San Diego, Calif.
U.S. Naval Preventive Medicine Unit 6, Pearl Harbor, Hawaii.
Ammunition Depot, Crane, Ind.
Naval Propellant Plant, Indian Head, Md.
Boston Naval Shipyard, Boston, Mass.
Portsmouth Naval Shipyard, Portsmouth, N.H.
Philadelphia Naval Shipyard, Philadelphia, Pa.
Norfolk Naval Shipyard, Portsmouth, Va.
Charleston Naval Shipyard, Naval Base, S.C.
San Francisco Bay Naval Shipyard, Vallejo, Calif.
Puget Sound Naval Shipyard, Bremerton, Wash.
Pearl Harbor Naval Shipyard, Pearl Harbor, Hawaii.
Naval Air Station, Jacksonville, Fla.
Naval Air Station, Pensacola, Fla.
Naval Air Station, Corpus Christi, Tex.
Naval Air Station, Norfolk, Va.
Naval Air Station, San Diego, Calif.
Naval Air Station, Alameda, Calif.
Naval Weapons Plant, Washington, D.C.

Naval Air Station, Quonset Point, R.I.
Naval Air Missile Test Center, Point Mugu, Calif.
Armed Forces Institute of Pathology, Washington, D.C.

In addition to the well-defined body of literature on occupational health and industrial hygiene, the Flight Surgeon has available to him the following periodical publications:

U.S. Navy Medical Newsletter, NAVMED 5088 (published biweekly).

Quarterly Occupational Health Hazards Reports, BUMED 6260.1 series.

Flight Surgeon's Newsletter, published by the Naval Aviation Safety Center, Norfolk, Va.

The discussion which follows in the remainder of this chapter emphasizes the more significant toxic substances found in the aviation environment and the hazards they produce. These have been selected as most important (although there are others of importance), based on two criteria: the frequency with which hazardous exposures to a substance may occur, and/or the severity of the hazard to health and performance.

AVIATION FUELS AND PROPELLANTS

A staggering amount of engine fuel is consumed yearly in the Navy, involving a considerable number of people in fuel-handling operations. This high exposure factor, coupled with a highly volatile, flammable, and toxic class of substances, makes the health and safety of ground and air personnel a matter of considerable concern. In view of this, the Flight Surgeon must be especially alert to the problems and hazards surrounding the handling and use of high-energy fuels. Fuels possessing potential toxicological problems in the aviation environment include gasoline and jet engine fuels, and the fuels used in missile operations.

Aviation Gasoline

The principal toxicological agents in aviation gasolines are the aliphatic and aromatic hydrocarbons. Other toxic substances are found in small quantity, such as tetraethyl lead and tricresyl phosphate. Typically these present no hazard unless the exposure is severe. For example, lead poisoning can occur when empty gasoline storage tanks are entered by cleaning personnel.

Gasoline vapors are heavier than air and are readily absorbed by the pulmonary epithelium. When the concentration of gasoline vapor in air is high, absorption by the lungs occurs quickly with symptoms manifested within minutes after inhalation. The maximal concentration of automobile gasoline vapor allowed by industries requiring daily exposure of personnel ranges from 0.05 to 0.1 percent (500–1,000 ppm), although symptoms have been manifested below 0.05 percent (Aero Medical Association, 1953). Since aviation gasolines are more toxic than automobile gasolines, the safe limits of concentration are below these levels. In view of the stringent performance requirements in flight, immediate detection of gasoline fumes is required. Fortunately, the maximum allowable concentration is above the human smell threshold; hence, the presence of odors is a clue that toxic concentrations are present.

Symptoms.—The volatile, aliphatic, saturated hydrocarbons are highly soluble in fat and are absorbed significantly in the lipoid constituents of the central nervous system and the blood corpuscles. Exposure to high concentrations of vapor in closed spaces results in systemic effects. Within minutes, dizziness, nausea, and headache occur. Signs of acute intoxication include excitement; disorientation; disorders of speech, vision, and hearing; cyanosis; and coma. Death may be the final result. Intoxication from gasoline fumes is similar to that resulting from drinking alcohol: 0.26 percent for a period of one hour (Aero Medical Association, 1953) will give rise to a drunken condition. Symptoms include giddiness, slurred speech, poor coordination, nausea, and a mild euphoria or combativeness.

When the skin is the site of exposure, which is frequently the case in ground handling operations, liquid gasoline produces local irritation. Repeated exposure may include burning, blistering, drying, and fissuring of the skin. Secondary infection is common. Liquid gasoline may also cause hyperemia of the conjunctiva and other eye disturbances, accompanied by redness and much tearing. Gasoline is not absorbed sufficiently by the skin to cause systemic effects.

Treatment.—When gasoline fumes are detected during flight, aircrew personnel should switch to 100 percent oxygen to avoid inhaling the fumes. This should be emphasized since gasoline odors are not unpleasant. The effectiveness of this simple procedure of 100 percent oxygen usage has been cited frequently. For example, in a recent case report on hydrocarbon vapor intoxication in aircraft (Davies, 1964), it was estimated that the concentration of fuel fumes in the cockpit was between 3,000 to 7,000 ppm causing intoxication in about seven minutes. The pilot had the presence of mind to switch to 100 percent oxygen, and successfully landed the aircraft.

Intense and prolonged exposure to fumes in confined spaces on the ground or deck, such as occurs in the cleaning of storage tanks, requires the wearing of self-contained breathing apparatus. To avoid contact with liquid gasoline, neoprene gloves and apron and nonsparking shoes should be worn. Goggles or face shields are required where splashing is likely.

Jet Engine Fuels

Jet engine fuels (JP-4, -5, and -6) contain more toxic aromatics and considerably more sulfur than does gasoline. Also, jet fuels do not contain tetraethyl lead. Being less volatile, lower concentrations might be expected to leak into the aircraft cabin. A comparison of the chemical composition of aviation fuels is shown in table 17-1.

Inhalation of vapors may result in mild narcotic effects similar to those of other hydrocarbon vapors. Due to this, concentrations approaching 1,000 ppm should be considered hazardous in the short-term situation. The TLV is 500 ppm. Problems associated with the handling of jet fuels are the same as those associated with the handling of aviation gasoline.

Symptoms and Remediation.—Same as for aviation gasoline.

Propellants

Rocket fuels, both solid and liquid, are combustible materials of high energy and reactivity containing powerful oxidizers. Many of these substances and their decomposition products are toxic to personnel. The handling requirements and use of these fuels present hazards to launch and fuel-handling crews.

The discussion of various rocket propellants which follows is based in part on material presented by Boysen (1961), Office of the Director of Defense Research and Engineering (1963), NAVMED 5035, Departments of the Army, Navy, and the Air Force (1954).

JATO Fuel.—JATO units use a propellant which is a mixture of potassium chlorate, engine oil, and tar. Recent developments have utilized nitro-

TABLE 17–1. *Chemical Composition and Other Properties of Aviation Fuels* †

Composition	Octane rating				JP–4	JP–5
	80	91 96	100 130	115 145		
Isopentane (percent)	15 to 20	15 to 20	15 to 20	15 to 20		
Alkylates (percent)	30 to 40	30 to 40	30 to 40	30 to 40		
Aromatics (benzene, toluene, xylene) (percent).	3 to 15	3 to 15	3 to 15	3 to 15	20	20
Straight run gasoline (paraffin series) (percent).	25 to 52	25 to 52	25 to 52	25 to 52	50	50
Olefins (C_5 and up) (percent).					5	5
Mercaptan sulfur (percent)					0.005‡	0.005‡
Total sulfur (percent)	0.05‡	0.05‡	0.05‡	0.05‡	0.4‡	0.4‡
Additives:						
Corrosion inhibitor (percent).					0.007‡	0.007‡
Tetraethyl lead (TEL) (ml, per U.S. gallon).	0.5	4.6	4.6	4.6		
Tricresylphosphate (ml, per U.S. gallon).				0.1 to 0.12		
Kerosene (percent)					30	30
Reid vapor pressure (psi)	5.5 to 7.0	5.5 to 7.0	5.5 to 7.0	5.5 to 7.0	2 to 3	0
Flashpoint (°F)	30 to 40				0	140
Freezing point (°F)	−76	−76	−76	−76	−76	−40
Explosive range (percent)					2 to 10	2 to 10
Dye	Red	Blue	Green	Purple		

† Octane ratings are those of military specifications (Mil–F–5572). Commercial fuels are essentially the same (ASTM D910–48T), i.e., red, 80/87; blue, 91/98; green, 100/130; purple, 115/145; plus an additional one, brown, 108/135. Tetraethyl lead (TEL) content is the same except: 91/98 contains 2.0 ml per gallon; 100/130, 3.0 ml per gallon; and 108/135, 3.0 ml per gallon.

‡These percentages are "by weight"; all others "by volume."

(After Boysen, 1961. Used by permission of the Williams & Wilkins Co. and the Reinhold Publishing Co.)

gen compounds which give rise to the highly toxic substance, nitrogen dioxide. From the position of JATO units on aircraft, and the frequency of use of this engine, it is highly unlikely that inflight exposure to the propellant or its exhaust gases will occur. However, JATO combustion products could prove a hazard to ground crews and care should be exercised.

Solid propellants possess powerful oxidizing agents which are potentially toxic. For example, potassium perchlorate ($KClO_4$) and ammonium perchlorate (NH_4ClO_4) cause severe skin burns on contact. The gases discharged during combustion consist primarily of sulphur dioxide (SO_2), hydrogen sulfide (H_2S), hydrogen chloride (HCl), and carbon monoxide (CO). Animals exposed to a mixture of these gases developed tracheitis, pneumonitis, pulmonary hemorrhage, and pulmonary edema. Severe conjunctivitis is noted in some animals.

Fuming Nitric Acid.—Red fuming nitric acid, one of the most widely used oxidizers, is a liquid containing dissolved oxides of nitrogen. Nitric oxide (NO) oxidizes rapidly into nitrogen dioxide (NO_2) which polymerizes to nitrogen tetroxide (N_2O_4). Inhaled fumes consist of approximately 70 percent N_2O_4 and 30 percent NO_2.

Fuming nitric acids are extremely corrosive oxidizing agents and cause severe skin and eye burns. Another serious hazard in handling fuming nitric acids is the inhalation of toxic vapors, especially nitrogen dioxide. Although nitric acid vapors are a pulmonary irritant, the primary toxic constituent of red fuming nitric acid is nitrogen dioxide. Its threshold limit value is 5 ppm (9 mg/m^3).

Symptoms.—The initial symptoms of poisoning are irritation of the eyes and throat, cough, tightness of the chest, and nausea. Hours later, severe symptoms begin suddenly, precipitated by exertion. These include coughing, a feeling of chest constriction, and cyanosis. In concentrations of 25 ppm the fumes irritate the eyes, nose, and throat. Concentrations of 60 to 150 ppm increase the irritation and produce a burning in the throat and chest. The possibility of pulmonary edema, cardiac failure, bronchopneumonia, and death depends on the time-dose relationship. Acute poisoning can result in pulmonary edema with the possibility of death within two days. Concentrations of 100 to 150 ppm are dangerous in exposures of one hour or less; very short exposures of 200 to 700 ppm may be fatal.

Nitric oxide is a cerebral depressant and can form methemoglobin. Nitrogen dioxide and nitrogen tetroxide are local irritants causing severe skin burns. Contact with the eyes may produce corneal changes and permanent impairment. Repeated exposure to low concentration levels may cause ulceration of the nose and mouth and chronic irritation of the respiratory tract. Bronchitis, bronchiectasis, and secondary pulmonary emphysema may develop.

Treatment.—Nitric acid, if spilled on the skin or in the eyes, must be removed immediately. For skin exposure, affected parts should be thoroughly washed with water, followed by washing and soaking with a weak sodium bicarbonate solution. If splashed into the eyes, liberal flushing with water for at least 15 minutes is recommended.

The individual exposed to the fumes should be immediately hospitalized and given absolute rest. He should be kept warm. Administering oxygen is often desirable. If breathing stops, artificial respiration with the aid of oxygen should be initiated. Care should be exercised in the use of sedatives because of the danger of synergistic respiratory depression. Venesection may be used to relieve heart overloading due to pulmonary edema, but complications may arise.

Safety Measures.—Vinyl-coated gloves and overboots made of rubber must always be worn when handling nitric acid. Protective body clothing, such as vinyl-coated fiberglass or polyethylene, must be worn. Fiberglass clothing impregnated with acid-resisting plastic, such as Teflon and Kel-F, is excellent. A hood is required to protect the head. Respiratory protection is required when exposure to nitrogen dioxide is prolonged at concentrations greater than the threshold value. An approved self-contained breathing apparatus provides the most reliable protection from fuming nitric acid. A catalog of stock respirators is presented in the Navy *Safety Equipment Manual* (NAVEXOS 422, 1960).

Boranes.—Various borane compounds (alkyl borane, diborane, pentaborane) are extremely toxic and hazardous to man. All may enter the body by inhalation, ingestion, or skin absorption. Contact of the liquid fuel with skin or eyes can cause severe local damage. The liquids can also penetrate skin to cause systemic effects. Vapors from these fuels are highly toxic and cause damage to the eyes, lungs, and upper respiratory tract as well as systemic effects. The systemic effects are complex and not well understood. Diborane produces pulmonary edema following inhalation. Prolonged exposure to low concentrations gives rise to lightheadedness, dizziness, fatigue, and muscular weakness. Pentaborane and alkyls primarily affect the central nervous system. High concentrations induce muscle spasms, tremors, and convulsions and, in some cases, depression, disorientation, and unconsciousness. Cardiovascular effects may occur, including changes in heart rate and blood pressure; in severe poisoning, blood pressure may be very high and later may fall to shock levels. Muscular or neuromuscular effects may contribute to weakness or unsteadiness. There may also be evidence of liver and kidney damage. Though the odor of these vapors is objectionable, odor should not be relied upon as sufficient warning. Recent TLVs for these fuels have been set as follows: diborane, 0.1 ppm; pentaborane, 0.01 ppm; and decaborane, 0.05 ppm.

Treatment.—Speed in removing liquid boranes is essential. Contaminated clothing must be removed and the skin thoroughly washed with soap and water. For HEF–3 or $HICAL_3$ alkyls, washing should be done with three percent aqueous ammonia. Large amounts of the ammonia solution are required as it forms a toxic compound when combined with the fuel. Eye contamination requires immediate flushing with water for at least 15 minutes. Anticonvulsant therapy or other sedation may be useful, but the use of drugs must consider the possibility of respiratory depression. The use of oxygen dissipates the symptoms for diborane and pentaborane.

Respiratory Protection.—Same as for fuming nitric acid.

Hydrazine.—Hydrazine is an extremely caustic fuming liquid used as a propellant in either the anhydrous or the hydrate form. Spilled on the skin or eyes, it causes severe local damage or burns and may cause dermatitis. It also can penetrate the skin to cause systemic effects similar to those produced when it is inhaled or swallowed. The inhaled vapor causes irritation of the eyes and respiratory tract, and also systemic effects. High concentrations are unbearably irritating so that the individual will withdraw if possible before suffering pulmonary damage. The threshold limit value is one ppm ($1.3 mg/m^3$).

Symptoms.—On short exposure, the systemic effects involve the central nervous system, with tremor being the most pronounced symptom. Exposure to higher concentrations results in convulsive seizures, severe hypoglycemia, and marked hemoconcentration resulting in circulatory shock. Death may occur. With repeated exposure, renal and hepatitic damage may occur as well as anemia.

Treatment.—The treatment is identical with that of the boranes. In the event of convulsions, quick-acting barbiturates should be administered, taking into consideration the possibility of synergistic depression of respiration.

Additional Propellants.—Some propellants make use of a mixture of hydrazine, aniline, and furfural alcohol. Evidence indicates that the physiopathologic effects of exposure would be primarily those of aniline. The main symptoms would be methemoglobinemia with resultant decrease in oxygen saturation.

A variety of other propellants are used in the military context. Table 17–2 presents the effects of exposure to a series of high-energy propellants. These data were compiled by the U.S. Air Force but are pertinent to naval air operations. Table 17–3 describes the characteristics of propellants relating to their handling and storage.

TABLE 17–2. *Effects of Exposure to Propellants*

Substance	Signs and symptoms	Liquid contact		Portals of entry			Personal protective devices
		Eyes	Skin	Skin absorption	Inhalation	Ingestion	
Nitric Acid Fuming HNO_3 and Nitric Acid Sulfuric Acid Mixture HNO_3 H_2SO_4 and N_2O_4.	Yellow staining of skin if skin contacts nitric acid. Concentrated solutions may cause death of tissue with clotting of the blood vessels in the immediate area, and subsequent scarring and deformity.	Flush with water for 15 minutes before treatment. May cause blindness.	Corrosive. Severe chemical burn. Wash contaminated area 15 minutes with water. Immediate medical attention is necessary.	Corrosive and irritating action is of more concern than absorption.	Extremely irritating. If oxides of nitrogen are inhaled, subsequent pulmonary edema may develop.	Drink large quantities of lukewarm water, warm soapy water, or milk. Induce vomiting. Immediate medical treatment is necessary. Membranes contacting the acid will be destroyed.	Boots, gloves, acid hood, protective suit. Gas mask required when cleaning spills. Self-contained breathing apparatus required when in confined area. Safety showers and eye washfountains required.

Hydrocarbon Fuels, JP, RP, Gasoline	"Naphtha jag" may occur if mild concentrations are inhaled (about 1,000 ppm). Characterized by a happy mental mood, similar to alcohol intoxication. Inhalation of very high concentration may result in acute intoxication characterized by nausea, headache, extreme weakness, convulsive movements, unconsciousness, cyanosis, death.	May cause hyperemia of the conjunctiva and other disturbances of the eye. Irritation of the eye is accompanied by redness and copious tear production.	Local effects with long contact including burning, blistering, drying and fissuring of the skin. Secondary infection is common.	May be toxic through penetration of the skin.	MAC is 500 ppm. Concentrations above 1,000 ppm may cause effects noted under signs and symptoms.	Slow absorption. Symptoms appear after a lapse of considerable time.	Neoprene gloves should be worn. Shoes of the nonsparking or nonstatic conductive type recommended. Neoprene-covered apron is useful. Goggles or face shields should be worn when splashing is likely. Wear self-contained breathing apparatus in confined spaces.

TABLE 17-2. *Effects of Exposure to Propellants*—Continued

Substance	Signs and symptoms	Liquid contact		Portals of entry			Personal protective devices
		Eyes	Skin	Skin absorption	Inhalation	Ingestion	
UDMH (or JP-X) NH_2 $(N(CH_3)_2$.	Immediate and violent irritation of the nose and throat, itching, burning, and swelling of the eyes. Temporary blindness may occur upon contact with the eyes. May produce liver and nerve poisoning.	Irritant. Flush immediately with water and continue for 15 minutes. Eyes should then be irrigated with a 3% boric acid solution.	Irritant. Immerse the affected area in magnesium sulphate solution, or flood with water. Water flushing should be followed by an application of boric acid paste.	High toxic rating. Readily absorbed.	High toxic rating. Irritates nose and throat. May cause lung damage.	High toxic rating.	Goggles, suitable fuel handler's gloves, full-coverage suits, and rubber boots required. Safety showers and eye washfountains mandatory.

Aniline $C_6H_5NH_2$.	Headache, nausea, ringing sensations in the ears, weakness in limbs, disturbances of sense of smell, sight, taste and hearing; rapid pulse and blueness of the skin (cyanosis). Breathing may become difficult. Chills, unconsciousness, death. A nerve and blood poison.	Flush immediately with water 10 to 15 minutes. Saturated boric acid solution may be used.	Mild sensitizer. May cause contact dermatitis. Wash with water and soak with saturated oxalic acid solution or a 5% acetic acid solution.	High toxic rating. Readily absorbed.	High toxic rating. Readily absorbed. Respirators required when atmospheric concentration exceeds 5 ppm.	High toxic rating. Readily absorbed.	Full-coverage suits, hoods, gloves, and boots of an approved type are required. Leather gloves and shoes are unsatisfactory. Safety showers and eye washfountains required.
Furfural alcohol C_4H_3-OCH_2OH.	Toxicity information is limited.	Redness, irritation, excessive tear production. Flush eyes with water.	Prolonged contact causes redness and irritation. Flush area with water.	Moderate toxic rating. Slight absorption.	Moderate toxic rating. Irritation to mucous membranes of the nose, throat, lungs. Hazard is slight at ordinary temperatures because of its low vapor pressure.	Toxic when ingested. Moderate toxic rating.	No special protective clothing required. Remove contaminated clothing and wash affected skin with water.

TABLE 17-2. *Effects of Exposure to Propellants*—Continued

Substance	Signs and symptoms	Liquid contact		Portals of entry			Personal protective devices
		Eyes	Skin	Skin absorption	Inhalation	Ingestion	
Ammonia anhydrous NH_3.	Eye irritation, conjunctivitis, swelling of the eyelids, irritation of nose and throat, coughing, dyspnea and vomiting. Severe injury to linings of respiratory passages may cause death.	Irritating to eyes. Corneal ulcers have been reported following liquid splashes. Wash with water for 15 minutes before treatment.	Irritating to skin, especially if it is moist.	Absorption is minor.	Some absorption might occur, but it is of secondary importance compared to its irritating effects.	Possibility of ingestion is remote. Irritating properties could be of primary concern.	Protection for eyes and respiratory tract required in concentration above 100 ppm.

Oxygen liquid O_2.	Liquid oxygen is nontoxic. Continued breathing of concentrated oxygen gas will act as a stimulant and in extreme cases may produce an effect similar to drunkenness. Liquid contact may destroy tissues.	Serious "burns" on contact. Flush eyes with water. May result in blindness.	Serious burns on contact. Immediate effects may be minimized by soaking the affected parts in water for a few minutes. Extensive burns require medical attention as soon as possible. Permanent disfigurement due to tissue damage may result.	No hazard from absorption.	No hazard	Likelihood of ingestion is remote. Absorption would be negligible but burning would be severe.	Asbestos degreased chrome leather gloves, leather footwear, head and face protection required. Flame-resistant clothing necessary. An apron of approved material necessary when liquid oxygen is handled in an open system. Safety showers required.
Hydrogen peroxide concentrated H_2O_2.	Stinging or burning sensation at site of contact. Area contacted is bleached.	Burns on contact with the eyes. Flush eyes with water for 15 minutes prior to treatment.	Burning sensation and bleaching of the affected area. Immerse affected area in magnesium sulfate solution or flood with water.	Negligible absorption thru skin.	Vapors are irritating to nose and throat.	If swallowed, encourage vomiting. Administer warm milk or sodium bicarbonate and obtain medical aid.	Safety shower and eye washfountains required. Full coverage of approved protective suits and hoods, gloves, goggles, and boots (polyvinylchloride coated) should be worn when handling or transferring hydrogen peroxide.

TABLE 17–2. *Effects of Exposure to Propellants*—Continued

Substance	Signs and symptoms	Liquid contact		Portals of entry			Personal protective devices
		Eyes	Skin	Skin absorption	Inhalation	Ingestion	
Methyl alcohol CH_3OH.	Nausea, vomiting, headache, dizziness, loss of muscle control, abdominal pain, blindness, convulsions, damage to the brain, and death. Symptoms are usually delayed for 9 to 36 hours after exposure.	Irritation. Flush immediately with water.	Slight irritation. Skin may become dry and cracked due to the solvent action of methyl alcohol.	Absorbed thru skin. Wash skin immediately if contaminated.	Slight irritation to mucous membranes. Readily absorbed. Odor detectable at 400 ppm.	Readily absorbed. Ten ml (⅓ ounce) may cause blindness. Less than ½ pint may cause death.	Neoprene, vinyl plastic polyethylene or rubber aprons worn over coveralls required. Protective gloves and boots desirable. Approved eye and face protection required. Respirators required if atmospheric concentrations exceed 200 ppm.

Ethyl alcohol C_2H_5OH.	Exposures to air concentration in excess of 1,000 ppm may cause headache irritation of the eyes, nose, and throat and if long continued, drowsiness and lassitude, loss of appetite, and inability to concentrate.	Flush immediately with cool, clear water.	Mild irritation. Skin may become dry and cracked due to the solvent action of the alcohol.	Slight toxic hazard.	Readily absorbed. 1,000 ppm exposure for 8 hours causes severe irritation to membranes of upper respiratory tract. Odor detectable at 2 ppm.	Readily absorbed.	Protective clothing not normally required in the handling, storage, loading or transfer of the alcohol. Eye washfountain required. Safety showers not required but desirable. Respirator required if atmospheric concentration exceeds 1,000 ppm.
Ethylene oxide C_2H_4O.	Irritation of eyes and respiratory tract. High concentrations cause vomiting, convulsions, and death with the lungs, liver, kidneys and adrenals showing evidence of injury.	Flush 15 minutes with water prior to medical treatment.	Skin "burns" similar to frostbite may occur through evaporation. Skin sensitization may occur. Immerse affected area in magnesium sulfate solution or flood with water.	Slight absorption. Evaporates extremely fast.	Irritation to entire respiratory tract. Severe high concentrations. Absorption is rapid.	Possibility of swallowing ethylene oxide is remote.	Eye protective devices should be worn at all times. Respirators should be used when the atmospheric concentrations exceed 50 ppm. Hand, foot, head, face, and body protection required when large quantities are handled.

TABLE 17-2. *Effects of Exposure to Propellants*—Continued

Substance	Signs and symptoms	Liquid contact		Portals of entry			Personal protective devices
		Eyes	Skin	Skin absorption	Inhalation	Ingestion	
N-Propyl nitrate $CH_3CH_2CH_2NO_3$.	Dizziness, abdominal cramps, vomiting, bloody diarrhea, weakness, convulsions and collapse. Small repeated doses may lead to weakness, general depression, headache and mental impairment.	Irritation. Flush with water for 15 minutes.	Prolonged or repeated contact causes skin injury ranging from yellow discoloration through marked irritation, swelling, and thickening, to severe damage. Wash affected area with soap and water.	Systemic effects have not been observed to result from skin contact.	May cause injury if inhaled. However, the vapor hazard is not great because of its low vapor pressure.	May cause injury if swallowed.	Foot, hand, body, head and face protection required. Gas masks (organic vapor type) should be readily available.

(U.S. Air Force *Flight Surgeon's Manual*, AF Manual 161-1.)

TABLE 17-3. *Characteristics of Propellants Relating to Hazards in Storage and Handling*

Storage compatibility*		Propellant	I.C.C. classification	Primary/secondary hazards	TLV** (ppm)	Remarks
Group	Division					
A	I	Fuming nitric acid	Corrosive liquid	Toxic/corrosive	5	Very toxic fumes; highly reactive with organic materials; liquid nitric acids extremely dangerous to body tissues.
		Nitrogen dioxide (nitrogen tetroxide).	Poison A	Toxic	5	
	II	Liquid oxygen	Nonflammable gas	Fire		Supports combustion vigorously; may rupture containers but does not itself burn; may form explosive mixtures with other materials.
	III	Fluorine	Flammable gas	Toxic/fire	0.1	Extremely reactive with most oxidizable materials; liquid extremely dangerous to body tissue.
		Chlorine trifluoride	Corrosive liquid	do	0.1	
		Perchloryl fluoride	Nonflammable	do	3	
		Hydrogen peroxide	Corrosive liquid	Fire/corrosive	1	Liquid dangerous to body tissues; can be made to decompose explosively; containers must be vented; vigorously supports combustion.

See footnotes at end of table.

Table 17-3. *Characteristics of Proellants Relating to Hazards in Storage and Handling*—Continued

Storage compatibility*		Propellant	I.C.C. classification	Primary/secondary hazards	TLV** (ppm)	Remarks
Group	Division					
C	I	Ethyl alcohol (denatured).	Flammable liquid	Fire/toxic	1,000	Flammable liquids; poisonous if taken internally.
		Methyl alcohol	do	do	200	
		Furfural alcohol	Unclassified	do	50	
		Avgas, JP series	Flammable liquid	do	500	
	II	Hydrazine	do	Toxic/fire	1	Very toxic and flammable materials; hydrazine vapors can be catalytically decomposed, with resulting fire or explosion.
		Methylhydrazine	do	do		
		UDMH	do	do	0.5	
		Aniline	Poison B	do	5	
		JP-4/UDMH(JP-X)	Unclassified	do		
	III	Anhydrous ammonia	Nonflammable gas	do	50	Liquefied gases that present a fire and toxic hazard; highly volatile.
		Methylacetylene	Flammable gas	Fire	1,000	
		Ethylene oxide	Flammable liquid	Fire/toxic	50	
		Hydrogen	Flammable gas	Fire		
	IV	Nitromethane	Flammable liquid	Toxic/fire	100	Shock-sensitive under certain conditions.
		n-propyl nitrate	Unclassified	do	25	
		n-propyl nitrate/ethyl nitrate.	Flammable liquid	do	(1)	

D		Diborane	Unclassified	Toxic/fire	0.1	Spontaneously flammable with air, extremely toxic. Diborane, a liquefied gas, must be kept at Dry-Ice temperature to maintain purity.
		Pentaborane	Flammable liquid	do	0.01	
		Alkyl boranes	Poison B	do	***	
E		Nitrogen	Nonflammable gas	Asphyxiant		Can replace air, causing asphyxiation; CO_2 stimulates respiration at moderate concentrations and causes narcosis at higher concentrations.
		Helium	do	do		
		Carbon dioxide	do	Toxic	5,000	

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[1] No value established.

*Materials in different groups shall never be stored together, with the exception of those in group E, which may be stored with materials in any other group. Propellants in different divisions within groups should be separated. If necessary, materials from different divisions within a group may be stored together in small quantities, provided that the individual characteristics are recognized. With respect to mixtures of these propellants, the most dangerous characteristics of the components will govern.

**Threshold limit value.

***Very low.

(From *Handling and Storage of Liquid Propellants*, Office of the Director of Defense Research and Engineering, 1963.)

CARBON MONOXIDE

Carbon monoxide (CO) is perhaps the most important gaseous poison, since more deaths have been attributed to it than to any other toxic gas. It is formed by the combustion of any carbon-containing material when an insufficient amount of oxygen is present. It is significant in the aviation environment since its deleterious effects on performance have been often reported. It is possible for exhaust-gas contaminants to enter the aircraft cabin through flaws in the ventilation system or the exhaust system. Since it is odorless, CO should be suspected whenever exhaust odors are detected. CO is also produced as a result of fire aboard an aircraft.

Carbon monoxide is absorbed through the lungs and combines with the hemoglobin, producing hypoxia; also hemoglobin partially saturated with CO does not release its oxygen normally, resulting in less oxygen being liberated to the tissues. The affinity of hemoglobin for carbon monoxide is from 250–300 times that for oxygen. This being the case, relatively low concentrations of carbon monoxide in the air can produce very high blood concentrations with time. For instance, 0.5 percent CO inhaled for 30 minutes while at rest will result in 45 percent carboxyhemoglobin concentration, which is sufficient to product total collapse.

The threshold limit value of CO is 100 ppm (0.01 percent by volume) for an eight-hour workday, and 400 ppm (0.04 percent by volume) for daily exposures of one hour or less. The allowable limits of CO contamination in naval aircraft are outlined in NAVAIRINST 3750.1 series. Figure 17–1 shows the effects of carbon monoxide in terms of concentration and exposure time. Table 17–4 indicates the physiological response to various concentrations of carbon monoxide.

Carbon monoxide is colorless and odorless and may cause the individual to collapse and lapse into unconsciousness with no warning. With the concentration of 0.04 percent CO in the air (sea level), the effects are headache and nausea after one or two hours. Unconsciousness appears within 30 minutes with a concentration of 0.2 to 0.25 percent. Concentrations of 0.60 to 0.65 percent may produce unconsciousness and the danger of death in 10 to 15 minutes. These effects may occur in less than three minutes when the concentration is about 1.30 percent. As little as 0.01 percent concentration can cause symptoms of poisoning if the gas is inhaled for a sufficient period of time. While the onset of symptoms depends on concentration of the gas,

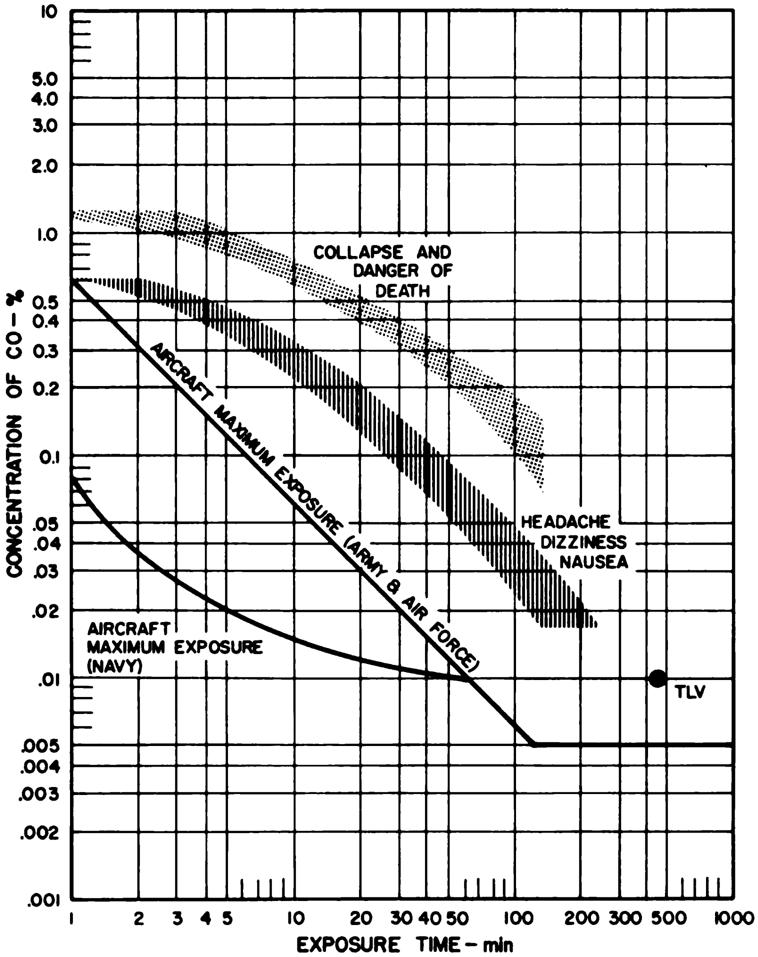

FIGURE 17–1. *The graph shows the effects of carbon monoxide on man as functions of concentration and exposure time. Milder effects are shown as a lightly shaded band of exposure times and concentrations, while dangerous or lethal times and concentrations are grouped in the heavily shaded band. The solid lines are the exposure limits set by the military services for aircraft. The point marked at 0.01% CO (100 ppm) and 480 minutes is the current Threshold Limit Value (TLV) for 8-hours-a-day exposure in industry.* (BACK, THOMAS, & PINKERTON, 1964; SOURCES: DEPARTMENT OF DEFENSE; HALDANE; HENDERSON & HAGGARD; & SAYERS ET AL.)

other factors such as individual differences and ambient environment conditions (wind, humidity, temperature) modify the effects (Fleming et al., 1960).

TABLE 17–4. *Physiological Response to Various Concentrations of Carbon Monoxide*

CO in air (volume percent)	PPM (volume)	Response
0.01	100	Concentration allowable for an exposure of several hours.
0.04–0.05	400–500	Concentration inhaled for one hour without appreciable effect.
0.06–0.07	600–700	Concentration causing just appreciable effects after one hour of exposure.
0.1–0.12	1,000–1,200	Concentration causing unpleasant but not dangerous symptoms after one hour of exposure.
0.15–0.2	1,500–2,000	Dangerous concentration for exposure of one hour.
0.4 and above	4,000 and above	Concentrations fatal in exposures of less than one hour.

(From Patty, 1963, based on the work of Henderson, et al., used by permission of John Wiley & Sons, Inc.)

Altitude and Carbon Monoxide

The toxicity of carbon monoxide increases with altitude. This is partly due to CO combining with more hemoglobin at the lower oxygen partial pressures existing at high altitude. In the absence of evidence of direct toxicity of carbon monoxide, it must be presumed that the toxic effect of CO occurs because of the extreme affinity of hemoglobin for CO, which causes a decrease in the oxygen saturation of the hemoglobin, resulting in relative hypoxia.

At 10,000 feet, the tracheal partial pressure of oxygen of the normal aviator would be about 100mm Hg, and the alveolar partial pressure of oxygen would be about 60mm Hg. Examination of the oxyhemoglobin dissociation curve shows that at 60mm partial pressure of oxygen and 38° C, the hemoglobin would be 89 percent saturated with oxygen.

A ten percent carboxyhemoglobin would result in an oxyhemoglobin saturation decrease amounting to ten percent times 89 percent or 8.9 percent, which would leave an effective oxygen-hemoglobin saturation of 80.1 percent. This does not take into account the change in the oxyhemoglobin dissociation curve caused by the changed pH resulting from the circulating carboxyhemoglobin. Changes would also occur in tissue oxygenation with time, due to respiratory adjustment. The effects of neither of these factors can be calculated but must be measured experimentally.

The net effect of the carbon monoxide-hemoglobin saturation of ten percent at altitude would be to cause onset of "anemic" hypoxic symptomatology in a shorter time and at a lower altitude than would be expected from the deceased partial pressure of oxygen alone. Figure 17–2 shows tolerance limits to CO at various altitudes as a function of exposure time.

Symptoms

The occurrence of symptoms is governed by the concentration of CO and exposure time, rate and depth of respiration, and altitude. Impairment of function due to CO exposure occurs earliest in the higher centers of the nervous system controlling cognitive and psychomotor abilities, and in the heart.

Concentrations of carbon monoxide-hemoglobin in the blood up to ten percent do not produce observable symptoms, although changes in the brightness threshold and night vision capability may be noted. Symptoms of mild intoxication (levels of carboxyhemoglobin below 20 percent) are vague and nonspecific, including subjective complaints such as mild frontal headache, general weakness, fatigue, and lassitude. With increasing blood saturation, the symptoms include nausea, vomiting, dyspnea, emotional disturbances, neuromuscular and joint pains, muscular cramps, tremor, anorexia, coughing, sweating, unconsciousness, collapse, and coma. The eyes may be dull and

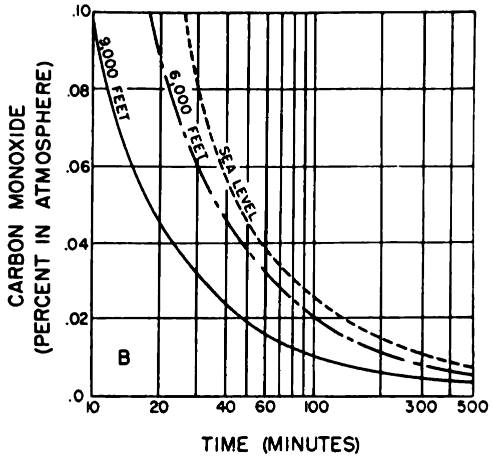

FIGURE 17–2. *Human tolerance limits for carbon monoxide.* (FROM Human Factors in Air Transportation BY R. A. MCFARLAND, COPYRIGHT 1953, MCGRAW-HILL. . . USED BY PERMISSION OF MCGRAW-HILL BOOK COMPANY.)

bulging, and with repeated exposure, visual, auditory, olfactory, and speech abnormalities may appear. In acute poisoning, there are few symptoms since unconsciousness occurs quickly. Recovery can occur even after the individual has been in a coma for several days. Pulmonary and nervous sequelae may result. Table 17-5 shows the progressive symptoms that occur with increasing concentrations of carboxyhemoglobin.

TABLE 17-5. *Effects of Carbon Monoxide Exposure*

Atmospheric concentration (ppm)	Percent blood COHb (80 percent saturation)	Time to 80 percent saturation (hours)	Symptoms
	0-10		None.
	10-20		Slight headache. Tight forehead.
200-300	20-30	5-6	Headache. Throbbing temples.
400-600	36-44	4-5	Headache, nausea, dizziness.
700-1,000	47-53	3-4	As above. Possible collapse.
1,100-1,500	55-60	1.5-3	Syncope, coma, convulsions.
1,600-2,000	61-64	1-1.5	As above. Decreased pulse and respiration. Possible death.
2,100-3,000	64-68	0.5-0.75	As above.
3,100-5,000	68-73	0.3-0.5	Weak pulse. Slow respiration. Probable death.
5,100-10,000	73-76	0.05-0.25	As above.

(From Patty, 1963, used by permission of John Wiley & Sons, Inc.)

CO poisoning may be distinguished from hypoxic hypoxia by a characteristic red flush on the face and by pink fingernails. In hypoxia, the fingernails and face become cyanotic.

Prophylaxis

When the presence of CO is suspected, either by the smell of exhaust gases or from symptoms of headache, nausea, or dizziness, turn off heaters and immediately don oxygen mask and select 100 percent oxygen to preclude breathing cockpit air.

Treatment

Administer 100 percent oxygen, if available. Apply artificial respiration. Symptomatic therapy may necessitate venesection, spinal puncture, and sedation; however, morphine and atropine sedation are contraindicated. With severe poisoning hypothermia may be of value in therapy.

Uptake of Carbon Monoxide

The rate at which the concentration of carboxyhemoglobin increases in the blood is due to a variety of factors. These include: the concentration of the gas, rate of lung ventilation, blood volume, hemoglobin concentration, and duration of exposure. A basic formula for achieving a rough calculation of the carbon monoxide level in the blood or in the air, when one is known, is:

$$\%\ \text{blood CO} = K \times \%\ \text{air CO} \times \text{time (in minutes)}$$

In this formula, the constant K compensates for differences in ventilation rate and has been established as:

$K=3$ (rest)
$K=5$ (light activity)
$K=7$ (heavy activity)
$K=11$ (heavy work)

Detection of Carbon Monoxide

NAVAIRINST 3750.1 series, entitled "Carbon Monoxide Contamination; Prevention of," outlines the responsibilities of all personnel in prevention of CO contamination. It states that "Flight Surgeons shall make checks for carbon monoxide whenever its presence is suspected and make blood determinations on personnel involved."

OZONE

Ozone inhalation, a hazard of high-altitude flight, is becoming an increasingly urgent problem due to the increases in the ceiling of military aircraft and the development of supersonic transports for civil aviation. Present-day aircraft are just reaching the lower boundary of the ozone layer. It appears that ozone inhalation is not a medical problem below 40,000 feet of altitude. A recent investigation (Young, Shaw, & Bates, 1962) detected 0.3 to 0.4 ppm of ozone in a DC-8 jet airliner flying between 27,000 and 39,000 feet of altitude. This exposure is well beyond the TLV of 0.1 ppm, and evidently no detectable injury was experienced by the passengers or crew. No evidence of injury would be expected since the exposure was of short duration. Whether the concentration was constant or intermittent was not determined, but the latter possibility seems more likely. Most of the ozone in the atmosphere is formed between altitudes of 50,000 and 140,000 feet with peak concentrations of 10 ppm (by volume) between 70,000 and 90,000 feet.

Ozone (O_3) is an unstable and corrosive gas formed by the action of ultraviolet light on oxygen and by electric discharge in oxygen. In theory, it is produced through dissociation of molecules by ultraviolet solar radiation below 2,450 angstroms and the recombination of free oxygen atoms and undissociated oxygen molecules with other particles in the air. The collision of the excited atoms, nondissociated oxygen molecules, and other gas molecules such as nitrogen or water vapor produce ozone according to the equation: O (excited state) $+$ O_2+M (third body) $\rightarrow$ O_3+M (excited state). The combination of free oxygen atoms and oxygen molecules takes place only when the surplus energy of the oxygen atoms is transferred to the third body. Direct recombination of the free oxygen atoms to form O_3 is not possible because of their excited state (Jaffe & Estes, 1963).

Actually two photochemical processes, involving ultraviolet and infrared solar radiations are involved, which provide a check and balance system to ozone production and give it a characteristic pattern of concentration in the atmosphere. Simultaneous with ozone formation, ozone molecules are destroyed by sunlight in the infrared wavelengths of less than 11,500 angstroms.

At altitudes above 350,000 feet the ultraviolet radiation is so intense that all oxygen molecules are dissociated into oxygen atoms. Also, the rarefied atmosphere precludes three-body collisions and no recombinations of oxygen atoms take place. Below this is a transition region where three-body collisions will occur and oxygen atoms recombine and form oxygen molecules. No ozone is formed because the ultraviolet radiation level is too great.

Below 250,000 feet, ozone is produced. With altitude decreases, the concentration of ozone increases, peaking at about 70,000 to 90,000 feet and then dropping off to a negligible quantity at sea level. Figure 17-3 shows the general form of the ozone distribution and its concentration in parts per million (volume).

The threshold limit value for ozone set by the American Conference of Government Industrial Hygienists is 0.1 ppm for an eight-hour day. It appears that a level of 0.2 to 0.3 ppm is reasonable for repeated short exposures in typical jet aircraft flights. For very short exposures, still higher concentrations are considered to be permissible (Jaffe & Estes, 1963). At low concentrations, ozone has the odor of new-mown hay; above 0.5 ppm (volume) it has an obnoxious, acrid odor.

Biological Effects

In acute exposures, ozone is a highly toxic and lethal substance. Chronic low-level exposures may result in severe systemic disease. It is a powerful oxidizing agent irritating the membranes of the lungs. Ozone produces hypoxia by interfering with pulmonary gas exchange. It apparently is not absorbed by the blood nor has it a direct effect on body tissues. Evidence (Scheel, Dobrogorski, Mountain, Svirbely, & Stokinger, 1959) indicates that ozone reacts with the proteins of lung tissue, irritating the cells and altering the permeability of the cell wall, resulting in pulmonary edema. Repeated inhalation produces such injuries as fibrosis of the lungs. Chronic exposure to ozone also affects visual performance. This was demonstrated in a study

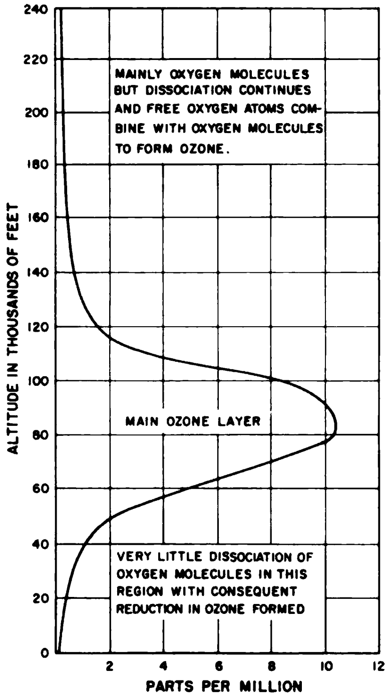

FIGURE 17–3. *Ozone concentration in parts per million, by volume* (ADAPTED FROM JAFFE & ESTES, 1963).

of 28 airline pilots who were experimentally exposed to ozone concentrations at altitudes expected to be flown by future transport aircraft. The subjects were exposed to ozone concentrations of 0.33, 0.58, and 0.83 ppm (by weight) for a total of slightly over 700 hours in periods of three and six hours. The results indicated significant changes in visual fields, night vision, lateral phoria, and convergence (Lagerwerff, Kane, & Thornburg, 1961). These effects of ozone indicate a significant safety hazard for high-altitude flight. Data from animal research indicate that a tolerance to the effects of ozone can be achieved by intermittent exposure to ozone or with premedications such as vitamin C. Increased susceptibility to ozone is brought about by physical exertion during exposure, alcohol, or respiratory infection. (See Jaffe & Estes, 1963, for a review of the literature.) A good deal of research has been conducted on ozone. Its toxic effects on animals have been described in the literature for over a century and there are recent studies describing human effects. The Flight Surgeon is urged to consult the various review articles in the literature for information; for example, see Thorpe (1954, 1955), Stokinger (1954), and Mittler et al. (1959).

The presence of ozone in aircraft cabins at altitudes above 40,000 feet is a distinct hazard to personnel. Fortunately, ozone is heat liable and can also be destroyed via filtering. The various filtering techniques include catalytic filters, charcoal filters, and molecular sieves. Some ozone is destroyed by heat when the ambient air is passed through compressors for pressurizing cabins. Unfortunately, present compressor design forces ambient air to move through too rapidly to take full advantage of adiabatic heating in reducing ozone content.

Symptoms

Above the TLV of 0.1 ppm, definite symptomatic effects occur. The most pronounced effects are pulmonary edema and hemorrhage, which may occur in one hour when exposed to 6 ppm. Acute exposure to 5 ppm for less than two hours results in coughing, throat irritation, chest pain, and dyspnea. Other effects include irritation of mucuous membranes and the ocular conjunctiva, substernal pressure, generalized body pain, and accelerated cardiac condition. Eventual coma and death may result. Chronic low-level concentrations may produce narcosis, headache, lethargy, and nausea. In accidental human exposures to high concentrations, central nervous system involvement has been demonstrated, and chest X-rays indicated increased bronchiovascular markings (see Lagerwerff, 1963). Table 17-6 outlines the effects of ozone in various concentrations (ppm).

One word of caution in diagnosis: ozone poisoning may not be suspected because of its similarity to other conditions showing pulmonary involvement. No special diagnostic tests are available.

Protective Measures

Flights above 40,000 feet in pressurized cabins (compression of ambient air) require the cabin to be fortified with oxygen. In Navy aircraft where the demand system furnishes 100 percent oxygen, ozone is not a problem, assuming proper functioning of oxygen equipment. On the ground, the characteristic odor of ozone can be detected before hazardous exposures have occurred and proper measures can be taken to safeguard personnel.

In the event of pulmonary edema, standard treatment for this condition is administered.

TABLE 17-6. *Effects of Ozone in Various Concentrations*

Observed effect	Concentration (ppm)
Threshold of odor, normal person	0.01-0.0015
Maximum allowable concentration	0.04.
Objectionable to all normal persons, irritates the nose and throat of most persons	0.10.
Disorders breathing, reduces oxygen consumption, shortens lives of guinea pigs	0.05-1.0.
Headache, respiratory irritation and possible coma	1-10.
Lethal to small animals in 2 hours	15-20.
Lethal in a few minutes	1,700.
Germicidal for airborne organisms	6,500.

(From Patty, 1963, based on Witheridge and Yaglou, used by permission of John Wiley & Sons, Inc.)

POLYMER FUMES

In aircraft, a large amount of electrical insulation is composed of a thermoplastic material called Teflon (tetrafluorethylene resin). This material is inert at normal temperatures and is nontoxic if ingested and nonirritating to the skin. In the event of fire, however, heat above 200° C causes Teflon to break down and certain pyrolysis products are liberated, consisting of strong acids (HF), ionizable fluorides, plus finely divided particulate matter. Above 200° C, inhalation of the products from heated polymer results in a condition known as polymer fume fever, or "the shakes," which is similar to an attack of the flu. Chills, lasting an hour or two, may develop after several hours, temperature may rise to 102° F, and a dry cough may develop. The main complaint is weakness and a feeling of exhaustion, sometimes accompanied by nausea or vomiting. The condition passes within 24 to 48 hours without complications. Usually, medication is not indicated. Bed rest and forced fluids are recommended and something to suppress the cough, if needed.

When Teflon is heated above 300° C, inhalation of the fumes may give rise to pulmonary edema. Treatment for exposure to the fumes follows the prescribed course for pulmonary edema. There is no evidence of any permanent or residual effects from inhaling the toxic fumes.

For protective measures, the oxygen mask is essential as a protection from fumes in the event of fire in the aircraft. In the ground complex, unprotected personnel should be evacuated from the immediate area of an electrical fire.

FIRE-EXTINGUISHING AGENTS

Another group of toxic agents that could prove hazardous to personnel are the fire-extinguishing agents. The inflight problem is primarily one of acute exposure to fumes released by these agents in the process of extinguishing a fire. Aircrew may come in contact with these agents following the use of portable extinguishers and in some instances may be exposed to fumes in the ventilation system from the action of automatic or semiautomatic fire-extinguishing systems aboard the aircraft. To a lesser extent, the inhalation of fumes from fire extinguishants may prove hazardous to ground support personnel.

Although the toxicity problems are not so severe in quality and frequency as with other toxic chemicals in the aviation environment, the Flight Surgeon must know the nature of the chemical reactions in order to provide treatment for exposure and also to familiarize flight personnel with the hazards in the use of these chemicals.

Fire-extinguishing agents in use today are of two chemical classes: (1) carbon dioxide, and (2) the halogenated hydrocarbons. The latter group comprises chlorobromomethane (CB) and dibromodifluoromethane (DB).

Carbon tetrachloride is also included but it is no longer recommended because of the extremely toxic nature of its thermal products. However, since it is in limited use, it is discussed briefly here.

Decisions on which chemicals to use are based on a number of factors. A prime consideration is the efficiency of the agent as an extinguishant. The halogenated hydrocarbons can be used to fight the major kinds of fire normally expected. These have been classified by the Navy as: Class A, combustible materials; Class B, flammable liquids; and Class C, electrical equipment. Carbon dioxide, however, is used only in Class B and Class C fires. Another factor is the toxicity of the agent. Carbon tetrachloride is the most toxic with a TLV of 25 ppm followed in order by DB, TLV of 100 ppm; CB, TLV of 400 ppm; carbon dioxide, TLV of 5,000 ppm. Allied with the toxicity of the agent is the toxicity of the thermal decomposition products released by the chemicals. For example, the pyrolized vapors of carbon tetrachloride, i.e., phosgene, are extremely toxic. Finally, the quantity of the chemical to be used is a determinant in the selection of the extinguishing agent. As an example, the toxicity of decomposed CB vapors is 15 times as great as that of the undecomposed vapors. But the quantity of agent needed is small and the time of application short; thus, the overall toxic hazard is slight.

Carbon Dioxide

As a fire extinguishant, CO_2 becomes a toxic hazard simply because large quantities of the chemical are used which release potent concentrations of vapor, usually in a confined space such as an aircraft cabin or a ship's hold.

A low concentration of carbon dioxide at sea level acts as a respiratory stimulant. Beyond this, inhalations of two to three percent bring on a feeling of discomfort, breathing becomes difficult, and the tidal volume is increased. The human can tolerate up to five percent concentrations for ten minutes. A concentration of about ten percent appears to be the maximum exposure for man, for at this level significant performance deterioration occurs. Concentrations above 20 percent can induce unconsciousness within several minutes.

Symptoms.—Initial acute exposures (less than two percent) of carbon dioxide will result in excitement and euphoria and, to some degree, improved efficiency. Rate and depth of breathing, heart rate, and blood pressure are increased. This is followed by drowsiness, headache, increasing difficulty in respiration, vertigo, dyspepsia, and muscular weakness. The individual exhibits incoordination and poor judgment. At concentrations beginning at ten percent, there is narcosis and collapse, and death may occur. The onset of symptomatology is slowed and the effects less marked if the increase in concentration occurs slowly, since compensatory mechanisms can act. One of the difficulties is that the individual, while aware of changes occurring, may be unable to assess correctly what is happening. The syndrome is similar to hypoxia but with the added stimulation of the respiratory center.

Figure 17-4 shows time-concentration curves of the toxicity of carbon dioxide. These data are based on the work of White (Aero Medical Association, 1953). The area below curve 1 represents the area of discomfort; between curves 1 and 2 is the area in which the individual may become confused, stuporous, or unconscious, or may function adequately; above curve 2 is the area of confusion; above curve 3 is the area of unconsciousness.

Prevention.—There is no special diagnostic test for CO_2. It is imperative that aircrew be indoctrinated in the hazards of carbon dioxide poisoning and that upon detecting any unusual vapors in the cabin area, immediately use 100 percent oxygen.

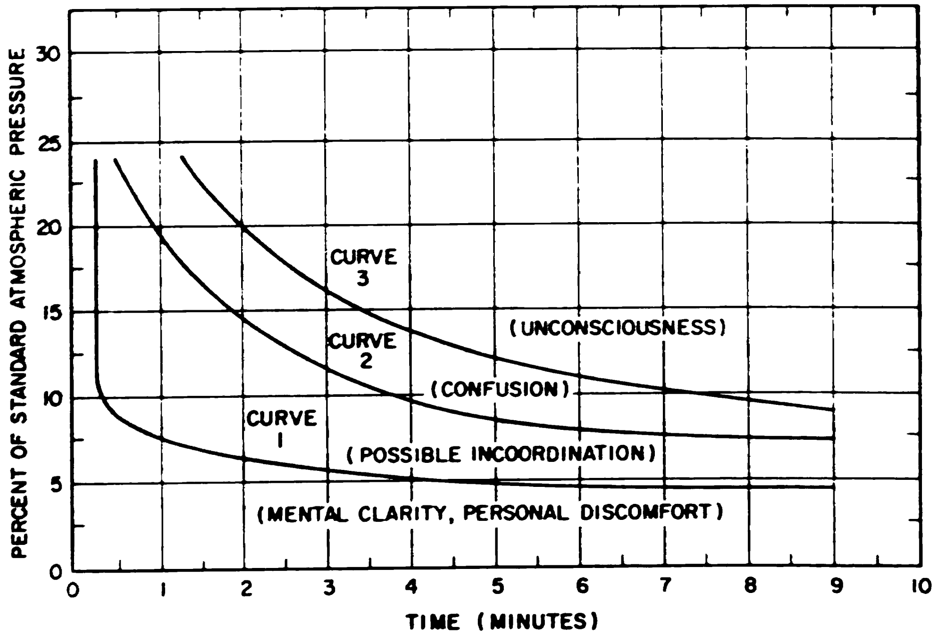

FIGURE 17-4. *Time-concentration curves for effects from carbon dioxide* (AERO MEDICAL ASSOCIATION, 1953, BASED OF THE WORK OF WHITE).

Since carbon dioxide is heavier than air, it accumulates in lower positions of enclosed spaces such as ships' holds. Normal air becomes diluted and the carbon dioxide acts as a simple asphyxiant. Ground support personnel must be made fully aware of this hazard so that preventive measures may be taken when required.

Chlorobromomethane (CB) and Dibromodifluoromethane (DB)

A feature of these halogenated hydrocarbons is that they are efficient fire extinguishants. While very toxic, the quantities necessary to stop a fire are relatively small; hence, the concentration of vapors tends to be low and the toxic hazard is lessened. They are more toxic per unit volume than carbon dioxide but considerably less than carbon tetrachloride. CB is slightly less toxic than DB (TLV of 40 ppm versus 1,000 ppm) in the undecomposed state. The decomposed vapors of CB (800° C) are about 15 times as toxic as in the undecomposed state.

Both CB and DB produce narcotic effects, the primary action being on the central nervous system. For both, the chronic toxicity is low, and adverse effects are not expected from repeated exposure below 0.1 percent.

Symptoms.—A major effect of halogenated hydrocarbons is lung irritation and damage. Effects of high concentration of CB include sneezing, coughing, respiratory distress, eye irritation, headache, nausea, and dizziness. Tremors and excitation have been noted with high concentrations of DB. These symptoms precede narcosis. In general, short-term inflight exposures are not so severe as to produce narcosis, but induce physiological aberrations of the kind described above with their resultant influences on aircrew performance.

Prevention.—Both CB and DB have distinct odors in high concentrations and CB is decidedly acrid. This provides warning of exposure and 100 percent oxygen should be used to avoid inhalation of fumes. In the ground environment, personnel should immediately evacuate the area.

Carbon Tetrachloride

Carbon tetrachloride (CCl_4), another halogenated hydrocarbon, is no longer approved as an aviation fire extinguishant because of its extreme toxicity to personnel. Since, however, some fire-extinguishing systems using this chemical may still be in existence, a brief account of CCl_4 hazards is included.

Under thermal decomposition, carbon tetrachloride produces an extremely toxic carbonyl; phosgene, which irritates the lungs and corrodes the lower respiratory tract. Sublethal exposures of this gas may permanently damage the respiratory system. Severe or prolonged exposure to carbon tetrachloride results in liver and kidney damage, with the latter being the usual cause of death.

Symptoms.—Inhalation of carbon tetrachloride vapors produces drowsiness, headache, nausea, vomiting, dizziness, anesthesia, and unconsciousness. A concentration of 0.1 percent produces symptoms in a matter of minutes. Inhalation of 0.6 percent concentration for five minutes results in unconsciousness and possibly death.

Figure 17–5 shows time-concentration curves of the toxicity of carbon tetrachloride. These data are based on the work of King (Aero Medical Association, 1953). The area between curves 1 and 2 is the time-concentration resulting in discomfort, nausea, headache, dizziness, and vomiting; the area above curve 2 is the condition for fainting, unconsciousness, and death.

Prevention.—The odor of carbon tetrachloride during thermal decomposition provides a warning of atmosphere contamination. For aircrew, the use of 100 percent oxygen is dictated. For ground personnel, adequate ventilation of work areas is required. Evacuation of the area is required under heavy concentrations.

SOLVENTS

Industrial solvents provide the most frequent and persistent toxicological problems in the aviation environment. As a group, solvents are the cause of the greatest number of man-hours lost in the performance of duty in ground jobs. A large number of chemically different solvents are used in the Navy today, designed for a variety of processes and operations. They are handled in some degree by many ground support personnel in such basic operations as degreasing, paint stripping, and painting, as well as in a number of specific operations. For example, one of the largest is metal degreasing involving both manual and machine processes. The manual activities include cold-dipping, brushing and wiping, and spraying. Machine operations include vapor, immersion, spray, and ultrasonic degreasing. The requirements and regulations governing these operations are described in

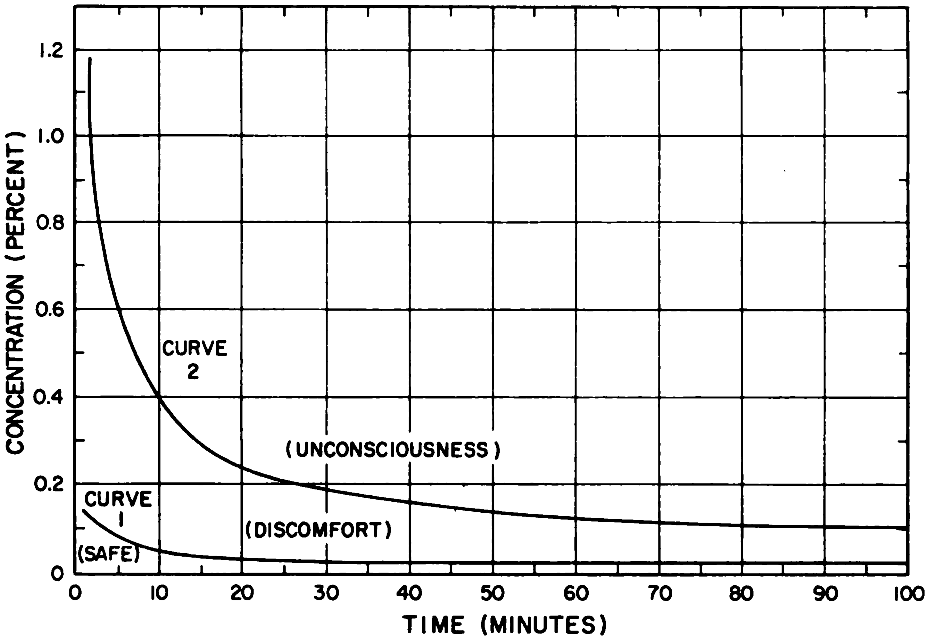

FIGURE 17-5. *Time-concentration curves for effects from carbon tetrachloride* (AERO MEDICAL ASSOCIATION, 1953, BASED ON THE WORK OF KING).

BUMED 6200 series of Instructions and Notices, and in Navy Civilian Personnel Instruction 792.

The hazards resulting from the use of solvents come about through inhalation and skin contact. Inhalation of vapors may produce anesthesia, and dermatitis is frequent since solvents remove oils from the skin. Skin contact may result in systemic effects and secondary infections may occur. Solvents are also eye irritants.

Since such a large number of solvents are cataloged in the Navy stocklist, it is difficult to discuss them all in a manner meaningful for this manual. Instead, this section will concentrate on the major classes of solvents and the particular toxic substances which are most frequently used in naval aviation.

The major classes of solvents important in the aviation environment (discussed here) are: (1) halogenated hydrocarbons, (2) aliphatic and aromatic hydrocarbons, (3) alcohols, and (4) ketones. Only the highlights of each class and the most important solvents in each class are presented. For greater detail the reader is referred to the specific sources in the voluminous literature on industrial hygiene and occupational medicine.

Halogenated Hydrocarbons

Halogenated hydrocarbons are used in large quantities in the Navy for removing grease and oil, paint stripping, and general cleaning, and to a lesser degree, for specific purposes. These solvents are health hazards, being dangerously narcotic, anesthetic, and poisonous agents when used improperly. Posioning may occur from inhalation or from skin contact, and more remotely, from ingestion. When absorbed into the body, certain of these agents may damage the liver and kidneys.

These solvents evaporate readily and build up unsafe conditions in the workroom environment unless adequate ventilation is provided. Most of these solvents, upon thermal decomposition, form extremely toxic materials.

Personnel who drink alcoholic beverages excessively are especially susceptible to physiological damage since the alcohol and the solvent mutually enhance each other's effect. As a rule of thumb, those halogenated hydrocarbons with a TLV less than 100 ppm are in a hazardous concentration when the odor is noticeable.

The halogenated hydrocarbons most frequently used in ground support operations are: trichloroethylene, perchloroethylene, methylene chloride, 1,1,1-trichloroethane (methyl chloroform), and freon.

Symptoms.—Acute inhalation of large concentrations of gas or vapor generally causes narcosis, evidenced by dizziness, headache, nausea, vomiting, mental confusion, and perhaps loss of consciousness. Continued exposure may result in coma and death. Removal from exposure is usually all that is necessary for recovery. However, tetrachloroethane (and carbon tetrachloride, which is no longer used as a solvent in the Navy) may cause extensive liver and kidney damage, with serious illness or a delayed death a possibility. Chronic exposures in excess of the TLV can result in serious illness from which recovery is difficult. The more toxic substances produce liver and kidney damage; the less toxic substances act on the central nervous system.

The inhalation of freons may produce mild irritation of the upper respiratory tract and mild central nervous system depression. Thermal decomposition and chlorine-containing freons may cause severe irritation of the eyes and throat.

Skin contact can result in dermatitis through defatting action on the skin.

In addition, solvents dry the skin causing cracks which may become infected. Many of the solvents can be absorbed through the skin with effects similar to those obtained from inhalation.

Methylene chloride is one of the least toxic of the halogenated hydrocarbons (500 ppm), while chloroform, trichloroethylene, and perchloroethylene are moderately toxic (100 ppm). Tetrachloroethane (rarely used in the Navy) is the most toxic of the halogenated hydrocarbons (5 ppm). The freons are mildly toxic (100–1,000 ppm). The vapors can be lethal because they may displace oxygen and cause suffocation. Table 17–7 presents a summary of the characteristics of halogenated hydrocarbon solvents used in naval aviation.

Treatment.—The victim must be removed from exposure and put at rest. If unconscious, artificial respiration and oxygen are recommended. Saturated clothing should be removed and liquids wiped from the skin. The individual should be kept warm. When eyes are contaminated, they should be flushed with large amounts of water. Adrenalin should not be used in any case of anesthesia because of possible ventricular fibrillation.

In the case of anesthesia produced by 1,1,1-trichloroethane, oxygen therapy may be used but epinephrine is contraindicated. Recovery from a nonfatal acute episode can be expected to be prompt and complete.

Prevention.—All halogenated hydrocarbons must be used with adequate ventilation. When vapor levels cannot be kept below the TLV, an air-supplied respirator should be used. Eye and skin contact must be prevented by the use of chemical goggles, impervious gloves, and protective clothing. Most halogenated hydrocarbons attack rubber so protective gear must be resistant to attack. Solvents or their vapors should not be exposed to high temperatures, open flames, or ultraviolet radiation.

Individuals known to be alcoholics or suffering from kidney, liver, cardiac, or neurological disease should not be placed at work where hazards of acute exposure or substantial repeated exposures exist.

Alcohols

The most prominently used solvents in this class are methyl alcohol, ethyl alcohol, and isopropyl alcohol. As a group, alcohols are not highly toxic. Ethyl and isopropyl are used in limited quantities for special cleaning jobs and in laboratory work. Methyl alcohol is the most hazardous. For this reason it is described in some detail here.

Table 17-7. *Characteristics of Halogenated Hydrocarbon Solvents*

Solvent	Phys. state at ord. temp.	Industrial uses	TLV (ppm)	Toxicity	
				Acute (short term, high exp.)	Chronic (long term, low exp.)
Chlorobenzene	Liquid	Solvent	75	Anesthetic effect, central nervous system depressant.	Liver and kidney damage.
Ortho-Dichlorobenzene (o-Dichlorobenzene).	do	Solvent, fumigant, insecticide.	50 (limit)	Definite organic injury involving liver and kidneys.	No apparent injury below the TLV.
Tetrabromomethane (acetylene tetrabromide).	do	Gauge fluid, solvent	5	Narcosis, coma—general central nervous system depressant.	Lung irritation, kidney and liver changes.
Methylene chloride	do	Paint remover, plastic solvent.	500	Narcosis, loss of equilibrium.	Slight skin and eye irritation.
Carbon tetrachloride	do	Solvent, fire extinguisher fluid.	10	Dizziness, nausea	Significant liver damage.
Ethylene dichloride (Dichloroethane).	do	Fumigant, solvent	50	Central nervous system depressant, liver and kidney change.	Skin absorption is significant. Liver, kidney, adrenal damage.
Methyl chloroform (Trichloroethane).	do	Degreasing solvent	350	Intoxication	Little or no physiological response.
Tetrachloroethane (acetylene tetrachloride).	do	Solvent, M-4 DANC-A CW decontamination & neutralization chemical.	5	Respiratory irritation and pulmonary damage, liver damage. Most toxic chlorinated hydrocarbon.	Significant skin absorption, liver damage.

Trichloroethylene (Trichlor).	.....do.........	Degreasing solvent..........	100............	Central nervous system depression, visual disturbance, nausea.	Affects nervous system.
Tetrachloroethylene (perchlorethylene, perchlor).	.....do.........	.....do....................	100............	Central nervous system depression, dizziness, incoordination.	Mild nausea, affects nervous system.
Freons[1]..................	Gas, liquid.....	Refrigerants, aerosol can propellants, solvents, fire-extinguishing agents.	100–1,000......	Most freons are relatively nontoxic. Decomposition products toxic.	

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[1] There are approximately 15 freons commercially available, ranging from liquids to gases. All contain fluorine and chlorine or bromine in various combinations. (Data from BUMEDINSTs 6260.12 and 6270.3 series.)

Methyl Alcohol.—Methyl alcohol is commonly used as a paint thinner, duplicator fluid, "canned heat," as a reagent in the laboratory, and as antifreeze. De-icing fluids and water-injection fluids in aircraft contain from 5 to 99 percent methyl alcohol. It becomes a deadly poison when vapors are inhaled in excessive concentrations or when taken internally. It is indistinguishable from grain alcohol by odors or sight and it cannot be rendered nonpoisonous. Methyl alcohol has been responsible for more deaths than any other poisonous substance used in the Navy. As little as four ounces taken internally causes death and two ounces or less can cause permanent, total blindness.

Symptoms.—With ingestion, acute gastrointestinal symptoms and central nervous system depression occur, including irritability, restlessness, labored respiration, and mental aberration. Various visual disturbances may appear, such as pain in the extrinsic musculature, congestion of the sclerae, optic neuritis, and retinitis. Death is usually due to respiratory or cardiac failure or to kidney injury.

Inhalation of very high concentrations may produce central nervous system depression and optic nerve damage. Lower concentrations may cause headache, dizziness, weakness, fatigue, and gastrointestinal or visual disturbances. The TLV is 200 ppm. Workers exposed to 400–500 ppm for short periods and up to 2,000 ppm for one-half hour or less show no detectable effects. Delayed death may be caused from 40,000 ppm during part of a working day.

Treatment.—For inhalation or skin contact, remove the individual from exposure, wash the affected skin areas, and irrigate eyes with water. Keep the individual quiet and institute artificial respiration if breathing has ceased.

Treatment of acute overexposure should consist of direct alkali treatment of acidosis when indicated.

A one percent solution of sodium bicarbonate is recommended for gastric lavage. Emetics, saliva cathartics, pilocarpine, diaphoretics, oxygen, saline infusions, stimulants, and spinal puncture may be indicated in therapy.

Ethyl alcohol presents no great toxicity problems with the exception of the symptoms of alcoholic intoxication when inhaled in high concentrations. It is absorbed, oxidized, and excreted quickly, which accounts for its toxicity in single acute doses. High concentrations produce headache, drowsiness, anorexia, and irritation of the eyes and respiratory tract. Absorption through the intact skin does occur but is not a toxicological problem. The recommended threshold limit is 1,000 ppm.

Aliphatic and Aromatic Hydrocarbons

The most frequently used solvents in this group are naphtha, Stoddard's solvent, toluene, and benzene (benzol). The features of the aliphatic compounds of naphtha and Stoddard's solvent are their low toxicity and their economic value of low cost per volume. They are, however, fire hazards.

Benzene, an aromatic hydrocarbon, is an excellent solvent for various gums, resins, fats, alkaloids, and rubber, and is a common ingredient in paint and varnish removers. It is highly toxic (TLV of 25 ppm). A single exposure of 3,000 ppm is endurable for one hour; 7,500 ppm is dangerous in 30 to 60 minutes. An exposure of 20,000 ppm is fatal within ten minutes. Absorption occurs chiefly by inhalation. High concentrations irritate the respiratory tract and produce narcosis. Repeated exposure to benzene may cause bone marrow damage, resulting in a decrease in the circulating white blood cells, platelets, and red blood cells. Many serious illnesses and fatalities have occurred in association with chronic exposures to benzene. A primary irritant type of dermatitis may result from repeated skin contact. Percutaneous absorption is considered insignificant.

Toluene is used as a solvent for a wide variety of gums, resins, and fats and as a diluent in cellulose ester lacquers. Although toluene resembles benzene in many of its physical and chemical properties, it does not produce the chronic blood diseases characteristic of benzene absorption. Toluene has a strong narcotic action, and effects are noted when workers' exposure exceeds the TLV of 200 ppm. Dermatitis may result from repeated skin contact. In experimental subjects, 600 ppm produced extreme fatigue, mental confusion, exhilaration, nausea, headache, and dizziness after three hours. Exposure to excessive concentrations may cause impairment in reaction time and in coordination.

For both benzene and toluene, first aid requires removal of the individual from the area of exposure. All contaminated clothing should be removed, the skin washed, and the eyes liberally bathed with water. Artificial respiration should be instituted if breathing has stopped.

Ketones

The most frequently used ketones are acetone and butanone (methyl ethyl ketone). They are primarily used as rubber solvents and rubber cement dilutors, and as solvents for resins and lacquers. These solvents are of low toxicity. Acetone is one of the least hazardous industrial organic solvents (TLV is 1,000 ppm) and is practically devoid of inhalation hazard. In very high concentrations it acts principally as a narcotic.

Butanone is capable of producing narcotic symptoms. The vapor is irritating (TLV is 250 ppm), hence, limits the possibility of exposure to high concentrations; 30,000 ppm is intolerable to man because of irritation of eyes and nasal passages. A concentration of 3,000 ppm is tolerable for only a short period of time.

These solvents can produce a dry, scaly, and fissured dermatitis after repeated exposure. High vapor concentrations will irritate the conjunctive and mucous membranes in the nose and throat. In high concentrations, narcosis is produced with symptoms of headache, nausea, vomiting, dizziness, incoordination, and unconsciousness.

Treatment for exposure does not usually require any special medical treatment. Extensive liquid contact requires removal of the contaminated clothing and the liberal flushing of underlying areas with water. In case of eye contact, flush liberally with water for at least ten minutes.

In the event of prolonged exposure to very high concentrations causing narcotic symptoms, remove the individual to fresh air and treat symptomatically.

Prevention procedures are the recommended safe practices for handling industrial solvents such as adequate ventilation, protective equipment, and respiratory devices, particularly for spraying operations.

EPOXY MATERIALS

Epoxy finishes which utilize amine hardeners are used to paint aircraft and various component parts. These materials are notorious producers of dermatitis and are sensitizing agents of such power as to require the permanent removal of the sensitized individual from the exposure. The amine hardener alone or mixed with the resin component prior to hardening is the principal offender.

In epoxy resin systems, the principal airborne materials are the vapors of various curing agents and dust from the machining of resins. Disability results from skin irritation and sensitivity to curing agents, hardeners, and solvents, and to uncured or incompletely cured resins. There is a potential hazard to certain types of amine curing agents.

Prevention

Proper ventilation is required to control the vapors produced while mixing resins and hardeners and to control the glass and epoxy particulates in any tooling or shaping operation.

Uncured resins and curing agents should be kept off the skin. Protective clothing is required, including the use of impervious aprons, sleeves, and gloves during mixing and application. Protective cream on exposed skin areas is recommended prior to using the material. In the event of accidental skin exposure, the material must be immediately removed with waterless cleanser, followed by a thorough washing with soap and water. Irrigate eyes promptly and for at least 15 minutes. Persons with active skin disease or history of pulmonary allergies should not handle epoxy materials in job operations.

The Flight Surgeon should take part in a program of education to inform the workers of possible hazards and to encourage good housekeeping practices here as well as in all ground support activities.

ADDITIONAL CHEMICAL SUBSTANCES

There are several additional classes of chemical agents important to the aviation environment. These agents, while toxic, possess the same hazard urgency as do the fire extinguishants. The probabilities of hazard to flight personnel are slight; nonetheless, it is important that the Flight Surgeon be aware of their characteristics and the effects produced. The agents are hydraulic fluid vapors, oil fumes, and anifreeze substances.

Lubricants and Hydraulic Fluids

The chemical structure of the lubricants and hydraulic fluids used in aviation are highly similar. They consist of 80–90 percent base oil that is either natural (petroleum, castor oil) or synthetic, plus chemical compounds which are added to achieve specific purposes. There are a number of hydraulic fluids and lubricants and they differ in their nature to the extent that the specific fluid must be identified prior to describing the hazard involved (Boysen, 1961). Table 17–8 shows the formulations of principal hydraulic fluids and lubricants.

Contaminants may enter the aircraft cabin as a result of leaks in the hydraulic system (pressure up to 3,000 psi) or from oil fumes entering due to ventilation system failure allowing air from the engine into the system. The possibility of exposure to these fluids or vapors, however, is slight.

The petroleum-base hydraulic fluids which are in general use in the Navy possess low volatility and the toxicity of the vapor is low.

TABLE 17–8. *Formulations of Typical Hydraulic Fluids and Lubricants*

Type	Chemical Composition	
	Compound	Percent
Hydraulic fluids:		
PRL-3169	Ethyl dibromobenzene	82.5
	Aroclor 1248	10.1
	Acryloid HF 55	3.9
	Mineral oil base stock	3.1
	Paranox 441	0.4
PRL-3209	Ethyl dibromobenzene	79.4
	Aroclor 1248	9.7
	Acryloid HF 55	3.6
	Calcium cetyl phenate (60 to 70 percent)	3.0
	Mineral oil base	2.9
	Tricresyl phosphate	1.0
	Paranox 441	0.4
PRL-3039	Di-secondary amyl sebacate	88.0
	Di-2-ethylhexyl sebacate	5.3
	Acryloid HF 25	5.2
	Tricresyl phosphate	1.0
	Phenothiazine	0.5
	Silicone DC 200	0.001
Lubricants:		
Mil-L-17535	Di-isoamyl adipate	96.65
	Barium petroleum sulfonate	3.00
	Phenothiazine	0.35
Mil-L-7808	Bis (2-ethylhexyl) sebacate	94.5
	Tricresyl phosphate	5.0
	Phenothiazine	0.5

(From Boysen, 1961.)

Castor oil-base fluids, rarely in use today, contain volatile constituents of butyl cellosolve, glycol derivatives, and alcohols. The vapors are toxic upon inhalation. The alcohols are a potent narcotic and together with butyl cellosolve produce irritation of the respiratory tract, headache, vertigo, irritation of the eyes, and impairment of vision.

Unfortunately, the natural oil-base fluids burn readily. This undesirable feature had led to a search for less flammable substances such as synthetic-base fluids. Synthetic-base fluids are less flammable but are toxic upon skin contact, passing readily through the dermal layer. This may produce pulmonary hyperemia and edema as well as degenerative changes in the liver, kidney, heart, and brain. Prolonged contact produces skin cracking and erythema. Accidental splashing will irritate the cornea and conjunctiva.

Basically, the hydraulic fluids possessing the greatest toxic hazard are those containing compounds of glycol, cellosolves, esters, or silicones. Additional toxic hazards result from the products of thermal decomposition. The toxicity of the halogenated hydrocarbons and the synthetic esters usually increases significantly under fire.

The symptoms of oil fume inhalation are similar to those found in carbon monoxide poisoning. These include irritation of eyes and upper respiratory tract, headache, nausea, and vomiting, and result from the products of oil breakdown, primarily acrolein and the aliphatic aldehydes. These substances are detectable in subtoxic concentrations by their irritant action and odor. When oil is heated above 500° F, the decomposition products are toxic, yielding such materials as keto acids, peroxides, aldehydes, carbon monoxide, and undecomposed oil particles. These irritate the nose and throat, cause eye watering, and otherwise make the individual uncomfortable.

Prevention.—In flight, inhalation of the noxious fumes and vapors can be prevented only by evacuating and/or ventilating the exposed area. The detection of any unusual vapors in the cabin requires immediate use of 100 percent oxygen. The problems of visibility and fire hazard are, of course, another matter.

It has been reported that ground support personnel have on occasion used hydraulic fluids as a suntan lotion. This is an extremely dangerous practice due to the systemic injuries that may be produced through skin contact.

Antifreeze Agents

Coolants and anti-icing fluids are common chemical compounds. The potential danger of exposure to these compounds is slight. In terms of quantity, very little anti-icing fluid is carried aboard an aircraft, and coolant fluids are not extensively used in the Navy. In this group most of the compounds are of a low order of toxicity with the exception of ethylene glycol (in coolant fluids), methyl alcohol, and ethyl alcohol.

Most often, the cause of inflight exposure to these agents is a break in a line which results in a fine spray of the substance under pressure. Coolant line ruptures often cause smoke in an aircraft cockpit, which is quite disconcerting to a pilot.

Ethylene glycol can become a hazard if it is liberated in a mist or spray under decreased atmospheric pressure. It has a low vapor pressure, hence, is unable to produce a vapor concentration high enough to be toxic even in an enclosed cabin. Ethylene glycol is a central nervous system depressant producing symptoms similar to ethyl alcohol intoxication. Inhalation may produce nystagmus, unconsciousness, and lymphocytosis. The TLV has not been established.

Thus far, the discussion has centered upon a number of toxic agents of importance in the aviation environment. These substances were selected for discussion in this chapter based on the criteria of frequency of encounter and the extent of the toxic hazard. By no means does this complete the catalog. There are a substantial number of toxic substances to which ground support personnel may be exposed. These may be so infrequently encountered or of such limited hazard that no discussion is presented. A summary of these agents and their health hazards is presented in table 17-9. Although the data have been compiled as the principal toxic agents found in Air Force operations, they are equally pertinent to naval air operations.

TABLE 17-9. *Control of Principal Toxic Agents*

Process where found	Toxic agent	Health hazards	Control measures
Aerorepair	Solvent dry cleaning Spec. PS-661a Small quantities other toxic agents such as toluene, acetone, amyl acetate, lead, ethyl alcohol, butyl alcohol, ethyl acetate, butyl acetate, petroleum naphtha, turpentine, carbon monoxide, caustic cleaners, greases	Dermatitis Pulmonary inflammation	General exhaust or good natural ventilation Local exhaust ventilation for cleaning inside of planes Protective hand creams Strict personal hygiene Good housekeeping Covered solvent containers Protective clothing
Assembled engine cleaning	Solvent dry cleaning Spec. PS-661a Rust inhibitor	Dermatitis Pulmonary irritation	Local exhaust ventilation Protective hand creams Protective clothing such as gloves, aprons, and boots

TABLE 17–9. *Control of Principal Toxic Agents*—Continued

Process where found	Toxic agent	Health hazards	Control measures
Battery	Lead Sulfuric acid Sulfur dioxide	Dermatitis Pulmonary irritation	General or local exhaust ventilation Personal protective clothing Isolation of process
Blacksmith	Carbon monoxide Sulfur gases Radiant heat Dust	Dermatitis Anoxemia Heat exhaustion	General and local exhaust ventilation Personal protective clothing Good housekeeping
Carburetor and ignition	Solvent dry cleaning Spec. PS–661a Trichloroethylene	Dermatitis Pulmonary irritation Renal damage Central nervous system damage	Local exhaust ventilation Covered solvent containers Protective hand creams
Electrical shop	Degreasers, solvents, oxides, selenium beryllium sulfate CCL_4	Dermatitis, lung and liver damage, malignant ulcers	Adequate ventilation both general and local, which may include booths, respirators, proper handling and disposal of fluorescent lighting tubes
Electroplating	Sodium cyanide Cadmium oxide Oxides of nitrogen Hydrofluoric acid Other chemicals which may be used are lead carbonate, copper sulfate, nickel sulfate, nickel chloride, phosphoric acid, acetic acid, caustic soda, chromic acid	Dermatitis (acid and caustic burns) Effects of hydrogen cyanide Edema of lungs Plumbism Conjunctivitis Chrome ulcers	Local exhaust ventilation (horizontal slot type preferable) General exhaust ventilation Isolation of process Protective cream Separate rooms and ventilation systems for acid solutions and cyanide solutions Personal protective clothing
Engine block test	Carbon monoxide Solvent drycleaning Spec. PS–661a Noise Oils, greases Gasoline, jet fuel	Dermatitis Anoxemia Temporary partial hearing losses Pulmonary irritation	Positive pressure ventilation of control room Local exhaust of oil return system Protective hand creams Protective ear plugs Proper design and location of block test buildings

TABLE 17-9. *Control of Principal Toxic Agents*—Continued

Process where found	Toxic agent	Health hazards	Control measures
Engine cleaning	Solvent drycleaning Spec. PS-661a Caustic cleaners removing (creosols and ortho-benzene)	Dermatitis Pulmonary irritation Possible CNS, kidney, and liver damage	Local exhaust ventilation Good natural ventilation Strict personal hygiene Protective creams Protective clothing
Engine disassembly	Solvent drycleaning Spec. PS-661a Oils, greases Other volatile solvents	Dermatitis	Good general or local exhaust ventilation Strict personal hygiene, protective clothing, protective creams
Fire protection and crash rescue	Fire extinguishants C.B. CO_2 carbon tetrachloride, heat, thermal decomposition, products of the extinguishants.	Acute narcosis, asphyxiation, liver and kidney damage, heat exhaustion, lung irritations	Proper training, ventilation of filling booths, use of respirators and masks
Foundry	Silica Carbon monoxide Metal fumes Excessive heat	Silicosis Anoxemia Heat exhaustion Metal fume fever	General and local exhaust ventilation Approved type dust respirators Good housekeeping Use of nonsilica parting compound
Fuel system repair shops	Solvents, gasoline, jet fuel, methyethylketone, ethylene dichloride, petron naphtha.	Dermatitis, narcotic effects, eye irritation, liver and kidney damage, acute anesthetic effects	Thorough indoctrination in precautionary measures. Use of protective equipment (hand creams, respirators, local exhaust ventilation).
Heat treating	Sodium cyanide Carbon monoxide	Dermatitis Effects of hydrogen cyanide Anoxemia	General and local exhaust ventilation (individual exhaust pipe for cyanide) Strict personal hygiene
Hydraulic	Solvent drycleaning Spec. PS-661a Hydraulic fluids	Dermatitis	Good general or local exhaust ventilation Protective hand creams Protective clothing
Hydraulic and pneudraulic shops	Solvents, alcohol hydraulic fluid	Dermatitis, aplastic anemia, atrophy of optic nerve, liver and kidney damage	Careful indoctrination of personnel; hand creams, respirators, adequate ventilation

TABLE 17-9. *Control of Principal Toxic Agents*—Continued

Process where found	Toxic agent	Health hazards	Control measures
Insect and rodent control section	Various economic poisons (insecticides and rodenticides). Solvents, kerosene	Skin irritants, dermatitis. systemic poisoning (liver necrosis, nephritis, convulsions, tremors from spinal cord and brain damage)	Skin protected by protective clothing. Safety goggles (chemical), respirators
Instrument repair	Solvent drycleaning Spec. PS-661a Naphtha Small quantities of miscellaneous organic solvents	Dermatitis	Local exhaust ventilation for solvent spray booth Good general ventilation Covered solvent containers Protective hand creams Protective clothing
Jet engine test stand and jet engine runup	Excessive noise exposure	Loss of hearing, damage to the cochlea, damage to the middle ear, rupture of the tympanic membrane. Fatigue, loss of muscular coordination. G.I. symptoms	Ear plugs, straightaway helmets, Models 372-8 and 372-9—Both helmets and plugs are recommended where the exposure is high and over a long period of time
Luminous dial painting and repair	Alpha and beta particles Gamma rays Radon gas Organic solvents	Radiation poisoning Dermatitis	Strict compliance with TB Med 232, "Radioactive Luminous Compounds Protective Measures"
Machine shops	Cutting oils and greases Miscellaneous dusts	Dermatitis	Strict personal hygiene Protective creams Local exhaust ventilation of dusty processes
Minor repairs	Solvent dry cleaning Spec. PS-661a Trichloroethylene Small quantities of miscellaneous organic solvents Caustic cleaning materials Oils and greases	Dermatitis Mucous membrane irritation CNS, liver, and kidney damage	Local and general exhaust ventilation Protective hand creams Personal hygiene Personal protective clothing and equipment

TABLE 17–9. *Control of Principal Toxic Agents*—Continued

Process where found	Toxic agent	Health hazards	Control measures
Paint and dope (brush and spray painting)	Paint thinner & dope containing misc. organic compounds, such as benzol, toluene, acetone, amyl acetate Ethyl or butyl alcohol Butyl acetate VM and P naphtha Turpentine Lead	Dermatitis Possible plumbism Blood changes and nervous symptoms	Exhaust ventilated paint spray booth Supply air respirator Chemical cartridge respirator Protective hand cream Isolation of process Closed solvent containers and separate storage of bulk paint and solvents
Parachute, leather, rubber and textile shop	Solvents, caustic cleaners, naphtha, methylethylketone, ethylene dichloride	Dermatitis, narcotic effects, acute anesthetic effects, eye irritations	Same as "Fuel System Repair"
Plexiglass	Small quantities ethylene dichloride	Dermatitis Anesthetic effect	General exhaust or good natural ventilation Covered solvent containers
POL hydrant refueling	Gasoline, J.P. fuels, Methanol	Dermatitis, plumbism, eye irritations, optic atrophy, acute anesthetic effects	Orientation of personnel in prescribed precautions, use of protective equipment such as chemical safety goggles, full face shield. Respirators may be required in high concentration. Protective clothing, deluge type shower—bubble type, eye washings, blankets.
Predock aircraft washings	Moisture, cold, kerosene and detergents	Chilling, dermatitis, nose, throat and eye irritations	Using ample protective clothing. Protective skin creams, respirators
Propeller	Solvent dry cleaning Spec. PS-661a Small amounts of oils, greases and trichloroethylene Paint remover	Dermatitis Possible narcotic effect and renal damage	Good general and local exhaust ventilation Protective hand creams Protective clothing

TABLE 17-9. *Control of Principal Toxic Agents*—Continued

Process where found	Toxic agent	Health hazards	Control measures
Radiator and tank oil coolers	Lead Compound carbon remover Caustic cleaning solution Solder and solder fluxes Greases and oils Solvent dry cleaning	Dermatitis Plumbism CNS, liver, and kidney damage	Local exhaust ventilation General ventilation Protective hand cream Protective clothing and equipment
Refilling fire extinguishers	Carbon tetrachloride Carbon dioxide Chlorobromomethane	Dermatitis Narcotic effect CNS, kidney, and liver damage	Local exhaust ventilated booth Good general ventilation Personal protective clothing and equipment
Rubber tank repair	Toluene Ethylene dichloride Small quantities of other materials, such as ethyl acetate, ethyl alcohol, methyl ethyl ketone, benzol, petroleum naphtha	Dermatitis Narcotic effect Nervous symptoms	General and local exhaust ventilation Protective hand creams Personal protective clothing and equipment
Sandblasting (abrasive cleaning)	Dust produced by pure silica sand organic abrasives, and steel shot	Dermatitis Pneumoconiosis (the higher the free silica content of the dust, the greater its hazard)	Proper maintenance of sandblast cabinets and equipment U.S. Bureau of Mines approved respiratory protective devices (supplied air helmets) Use of nonsilicous abrasives when possible
Sewage disposal plant	Methane, chlorine, H_2S and infections of skin and G.I. tract	Asphyxiations, lung irritations, dermatitis, conjunctivitis, and anemia	Training—Use of respirators or gas masks and immunization
Sheet metal shops	Deficiency of illumination, noise, eye strain, hearing loss. Source—mechanical injury	Dermatitis, injury	Properly designed workroom, protective clothing, frequent washings, ear defenders

TABLE 17-9. *Control of Principal Toxic Agents*—Continued

Process where found	Toxic agent	Health hazards	Control measures
Spark plug cleaning	Pure silica sand Solvent drycleaning Spec. PS-661a Trichloroethylene	Dermatitis Silicosis Narcotic effect CNS and renal damage	Local exhaust and general ventilation Protective hand creams Personal protective clothing and equipment
Vapor degreasing	Trichloroethylene Perchloroethylene	Narcotic effect CNS, liver, and kidney damage	Proper operation of vapor degreasers Locate degreasers away from drafts Keep degreasers covered when not in use Local exhaust ventilation when other corrective measures fail Supply air respirators and personal protective clothing when cleaning degreasers
Water plant	Chlorine gas, lime, soda ash, fluorides	Lung, nose, eye irritations, dental flurosis, caustic effects on skin	Use of respirators, gas masks. and good personal hygiene
Welding	Radiant energy Metal oxides including lead, iron, cadmium Gaseous decomposition products of red coatings Other toxic substances in small quantities, such as oxides of nitrogen, fluorides, and carbon monoxide	Dermatitis Flash burns of eyes Metal fume fever Heat exhaustion Edema of lungs Anoxemia	Protective helmet, shield, gloves, and apron (electric weld) Isolation of process General ventilation Local exhaust ventilation (always when producing cadmium or lead fumes) Portable or permanent black shield to protect adjacent workers
Woodworking shops	Wood, dust, glue, mechanical injury (dermatitis allergic reactions)	Poor illumination and safety devices. Toxemia from phenolic resins and paint solvents	Good ventilation and adequate lighting. Little or no contact with the skin. By using gloves, aprons, and respirators, and adequate hand washing facilities

TABLE 17–9. *Control of Principal Toxic Agents*—Continued

Process where found	Toxic agent	Health hazards	Control measures
X-ray and ionizing radiation	Ionizing radiation	Acute and chronic radiation sickness. Beta burns, adverse effect on the bones, lungs and blood, gonads, skin and mucous membranes. a. Skin—brittleness and ridging of the nails. b. Hands—increased susceptibility to chafing. c. Blunting or leveling of the finger ridges. d. Dryness and epilation. e. Changes in nail fold capillaries in the way of disordered pattern. f. Eyes—cataracts from neutrons.	Control of time of exposure; provision of shielding, respirators, etc. Use of distance to maintain total radiation dose below the permissible levels

(From U.S. Air Force *Flight Surgeon's Manual*, AF Manual 161–1.)

OXYGEN CONTAMINATION

A problem affecting the performance of flight crews concerns the contamination of oxygen. Objectionable odors in oxygen breathing systems using compressed gaseous oxygen or liquid oxygen have often been reported by aviators. A frequently reported odor is that of "rotten eggs" (due to mercaptan compound). While not present in toxic concentrations, this kind of odor quickly produces nausea and perhaps vomiting. In situations other than accidental or gross contamination, analysis of liquid oxygen has indicated the presence of small amounts of a number of contaminants. These include, water vapor, methane, carbon dioxide, acetylene, ethylene, nitrous oxide, traces of high hydrocarbons, and traces of unidentified contaminants (Bartlett & Phillips, 1961). Complaints of oxygen tank odors have also been attributed to the solvent, trichloroethylene, used in cleaning the cylinders.

Evidence to date indicates that the contaminants either singly or in combination never reach levels of concentration that are toxic to man. Most often the odors are neither offensive nor disagreeable, as indicated by such descriptive terms as stale, sweet, cool, fresh, or pleasant and unpleasant. Yet distinct symptoms have been reported including headache, sickness, nausea, vomiting, and in some instances, disorientation. The problem appears to be psychological rather than physical. In flight, the aviator is more concerned about breathing and is apprehensive about his oxygen source. Any odor detected becomes exaggerated. This stress-induced apprehension may lead to hyperventilation with its attendant real symptoms.

Mechanism of Contaminant Effect

There appear to be two ways in which symptoms are produced. One possibility is that lengthy exposure to a contaminant during flight could have an accumulative effect to make the individual aware of contamination. Man is able to sense extremely small concentrations of odors. For example, infinitesimally small concentrations of mercaptan are detectable (one part in 30 billion). Another possibility is that during the breathing of pure oxygen, an odor develops due to a momentary "surge" of a contaminant in the system. In liquid oxygen the contaminants may not be distributed homogeneously but may be concentrated in discrete particles. These crystals could melt in the converter and provide the individual with a temporary but relatively good whiff of an odor. This explanation may be more reasonable due to man's ability to adapt rapidly to a given odor (see Bartlett & Phillips, 1961).

It appears that odors are not identifiable per se: the awareness of the presence of a contaminant is based on the detection of a noticeable difference between the immediate odor and some previous reference odor. It is this difference that is acted upon and under the conditions of flight this low concentration contaminant may be perceived psychologically as toxic.

Dealing with the Problem of Oxygen Contamination

The frequency with which aviators develop symptoms of illness to nontoxic concentrations of "bad oxygen" indicates strongly the need for the Flight Surgeon to indoctrinate flight crews on the nature of oxygen contaminants. Such a program for providing information on the physiology of olfaction and the psychic reactions to oxygen contamination has been tried on a limited scale in the Navy with gratifying results (Bartlett, Phillips, & Couch, 1962). The psychological aspect is the most important feature since the individual's responses are exaggerated when viewed in terms of the actual concentration of contaminants. Evidence indicates that the odors in the system are not distinguishable. The individual is able only to detect a change in oxygen odor. Bartlett & Phillips, (1961) demonstrated that laboratory subjects were consistently able to differentiate contaminated samples of oxygen from pure oxygen but were not able to indicate which was which with any certainty.

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CHAPTER 18

Aircraft Accident Procedures and Investigation

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INTRODUCTION

Each accident involving Navy aircraft exacts a tremendous price in terms of the loss of highly trained personnel and expensive equipment. For this reason, Navy investigative efforts are designed to isolate the basic cause of each aircraft accident. When these causes are grouped, it is found that the human error category accounts for well over half of all naval aviation accidents. Human error, as used here, refers primarily to pilot error but includes also errors of other personnel such as tower controllers, GCA personnel, and supervisory and maintenance personnel.

The remaining aircraft accidents are attributed to system or material failure, miscellaneous, and "cause undetermined." Materiel failure accidents are investigated with extreme thoroughness; human error accidents, less so. This is simply because the investigative processes dealing with materiel problems are more advanced than those dealing with human factors. Once it has been established that an accident was caused by an improper response on the part of the pilot, it is extremely difficult, and often impossible, to go one step beyond and identify the specific characteristics of the pilot which made him behave as he did. Within recent years, however, the trend has been toward more sophisticated investigations of human error accidents, employing specialized techniques from fields such as aviation pathology and human factors engineering. Future years should provide more insight into the basic laws of human behavior which operate to produce these human error accidents.

It is the responsibility of the Flight Surgeon to see that proper evaluation is made of all human factors which may be involved in an aircraft accident. By virtue of his specialized training in the field of medicine, the Flight Surgeon possesses skills required to unravel the sequence of events occurring in an accident. A proper evaluation of the human factors requires that the Flight Surgeon have an understanding of the areas of forensic pathology, aircraft equipment design, safety and survival equipment design, psychology, physiology, and also that he be skilled in such matters as the interrogation of witnesses. (See app. 18–1, Interview Procedures.)

The indispensable factors in successfully dealing with an aircraft accident are planning and training. Unit and Medical Department Crash Orders form the nucleus around which preaccident plans revolve. Effective implementation of these plans is based on the cooperative efforts of well-trained and -disciplined personnel.

PREACCIDENT PLAN

A unit's preaccident plan is implemented in the event of aircraft accident. It is a document containing detailed instructions for those personnel assigned specific duties in handling accidents. Because of the wide variety of aviation units in the Navy (i.e., air stations, aircraft carriers, fighter squadrons, patrol squadrons, helicopter squadrons, etc.), there are marked differences among preaccident plans but all have certain common elements. These include:

1. A crash alarm system
2. Crash and rescue operations
3. Preservation of wreckage
4. Security and salvage
5. Public relations
6. Photography
7. Administrative procedures

As a general rule, the preaccident plan provides broad guidelines for medical personnel, which are further amplified in Medical Department Instructions. Items which should appear in the Medical Instruction are:

1. Personnel assignment
2. Communications.
3. Medical equipment and supplies
4. Disposition of the injured and deceased
5. Photographic assistance
6. Laboratory facilities
7. Preparation and shipment of tissue specimens
8. Administrative requirements (see app. 18–2 for General Death Procedures)

THE ACCIDENT

(The following outline refers primarily to shore-based activities and should be modified as necessary for shipboard and other applications.)

Upon notification of an accident, the Flight Surgeon should proceed immediately to the emergency vehicle. In most cases, this will be a field ambulance manned by hospital corpsmen assigned this duty. The vehicle should be equipped with an adequate emergency bag, intravenous fluids, resuscitator, oxygen bottles, splints, blankets, litters, remains pouches, crash investigation kit (which should include a 35mm camera), and special gear appropriate to the local geographic area (see app. 18–3). These items should be inventoried daily to insure that they are on hand and that perishable and sterile materials are replaced at appropriate intervals. Provision for rapid transfer of items to SAR aircraft is essential, and it is equally important that all flight gear required for such aircraft be readily available.

The emergency vehicle departs for the crash location as ordered by Operations. At the accident scene, rescue of the injured and protection of other persons in the area takes precedence over all other activity. The senior officer who first arrives is responsible for direction of military personnel and establishment of appropriate cooperation with civilian agencies. Qualified crash and rescue personnel assume this responsibility upon arrival at the scene.

On reaching the scene of an accident, the Flight Surgeon's first duty is to provide medical assistance to the injured. He should supervise their removal from the aircraft as well as their subsequent handling and treatment so as to avoid additional injury. If the wreckage is burning, medical personnel should render such aid as they can without interfering with the crash crew. Because of their special training and protective gear, crash crews are best equipped to remove individuals from burning aircraft.

The Flight Surgeon is responsible for rapidly evaluating the casualties and promptly notifying the receiving medical activity as to the number of patients, types and extent of injuries, method of transportation, and approximate time of arrival. He should also determine the best method of transportation for each individual and secure it insofar as is feasible.

When it is clear that a person has suffered fatal injury, his remains *must not be moved* unless it appears that additional physical alteration of the body may be incurred. There are two principal reasons for this:

1. Unless the accident occurs at a site where there is exclusive Federal jurisdiction or where prior agreement has been made with appropriate local authorities for removal of a body under specified circumstances, remains cannot be legally moved until authorized by a representative of the local government in authority. In most situations, the authorized local official is a medical examiner, coroner, or law enforcement agent.

2. Movement of bodies prior to examination by a trained medical investigator can destroy information of extreme value to the investigation. *It is imperative that crash crews, local fire departments, law enforcement personnel, rescue squads, and disaster emergency crews leave the fatally injured in place, when possible, until the Flight Surgeon completes his examination.*

The most effective way to insure that this is done is for the Flight Surgeon to meet with these groups periodically and explain to them the necessity for leaving the scene as intact as possible. They should be instructed to cover remains but otherwise leave them undisturbed. Under conditions where it is necessary to move a body, the investigator must carefully interrogate all personnel who took part in the removal.

THE INVESTIGATION

The basic Instruction which covers the investigation of each Navy aircraft accident, incident, or ground accident is OPNAVINST 3750.6 series. This Instruction covers in detail the requirements for the investigation, reporting procedures, control over accident data, and responsibilities for the foregoing.

For all aircraft accidents (except for those in which there is no greater damage than "substantial" and the cause is "simple and well defined") and those incidents and ground accidents which result in fatal, critical, serious, or severe injury and/or destruction of a Navy aircraft, the full Aircraft Accident Board shall consist of at least four officers, of whom two must be experienced Naval Aviators. This board is normally appointed by the reporting custodian (the activity to which the aircraft is assigned) but in some circumstances may be appointed by higher authority. The composition of the board is as follows:

1. The senior member shall be a Naval Aviator with wide experience or other qualifications warranting such an appointment and he must be senior to the pilot whose performance may come under investigation.
2. One member should be an officer well qualified in aircraft maintenance.
3. One member shall be a Flight Surgeon (Medical Officer in the event a Flight Surgeon is not available).
4. The fourth member shall be the Aviation Safety Officer.

For those accidents excepted above and for incidents and ground accidents not resulting in fatal, critical, serious, or severe injury and/or destruction of a Navy aircraft, the investigating board may consist of only one officer, who need not be senior to the pilot except in case of an aircraft accident. A Flight Surgeon shall be an additional member when circumstances require the submission of a Medical Officer's Report or when aeromedical factors are involved or suspected.

The sole purpose of these investigations is to determine all the contributing factors involved in Navy aircraft mishaps and to obtain information which can be used as a basis for corrective action. The proceedings and reports are considered privileged information and will not be used in any legal or punitive action.

Once the appointing order is issued, the board meets at the order of the senior member and determines the course of action which will be taken for each phase of the investigation. Subsequent progress meetings are held as deemed necessary.

The Flight Surgeon must actively participate in the deliberations of the board. His findings should be presented to the other members of the board in a manner that provides them a clear understanding of the significance of any aeromedical factors uncovered. He must be prepared to clarify any areas of misunderstanding which may arise in this phase of the investigation.

When the investigation has been completed, the board prepares the Aircraft Accident Report for submission to appropriate activities. The Medical Officer's Report forms a part of the AAR. These reports are forwarded to various activities for review. Among the reviewers is the Naval Aviation Safety Center (NASC). NASC carefully examines each of these documents and obtains information for coding and data processing. Information from these reports serves as the basis for studying accident causes and leads to elimination of hazard areas.

Medical Requirements

All pilots and aircrewmembers must have a medical examination following an accident and it should be accomplished as soon as practicable. The nature of the mishap and the condition of the individual determine the extent and depth of the examination. The professional judgment of the Flight Surgeon thus becomes the determining factor. The objective of this examination, over and above routine treatment, is to determine the aeromedical factors involved in the mishap and to make recommendations based on these findings.

The Flight Surgeon, as leader of the medical portion of the investigation, is responsible for examining the pilot's sociologic, psychologic, and physiologic background for factors which may have caused or contributed to the accident as well as for investigating the adequacy and functioning of safety and survival equipment in all phases of the mishap. He must understand what pathologic and/or toxicologic processes may have existed prior to the crash or were initiated by it. He must attempt to correlate injuries suffered by the aviator and agents causing them, whether they be instrument panel, flying missiles, control knobs, or anything else, in order to provide information on improvements in personal equipment and aircraft design which can decrease the injury potential of the man-machine system. In short, the Flight Surgeon must be capable of conducting an in-depth analysis covering a divergent number of complex disciplines.

There are two broad phases in the medical investigation:

The first is reconstruction of the accident from onset of the original problem through all events which ensued until recovery efforts were completed.

The second is the background investigation which includes gathering all the material on the pilot's background which may have played a causative or contributory role in the accident.

In accidents where there are survivors, obtaining the above information is, as a general rule, not too difficult. In the absence of survivors, the job is more difficult but certainly not insurmountable. Since statistically the largest number of accidents fall into the "pilot error" category, this is a most important part of the investigation.

The background study should develop a complete picture of the pilot's sociologic, psychologic, and physiologic structure and how these parameters may be related to the accident.

Chapter 10 of the *Handbook for Aircraft Accident Investigators,* NAVWEPS 00-50T-67, and Section K, Medical Supplement of OPNAVINST 3750.6 should be used as guides in this effort.

The Field Investigation

The Flight Surgeon should attempt to establish firmly in his mind the flight profile of the aircraft and the sequence of events which occurred during the crash. In the case of fatal accidents, physical evidence, the opinions of those experienced in aviation, and the testimony of reliable witnesses are the principal sources for such information.

Each portion of the wreckage should be meticulously examined for gross and trace evidence. Examples of such findings are blood spatters, tissue fragments, pieces of personal equipment, and marks produced by a particular item of protective gear (e.g., paint marks from helmets, etc.). The wreckage should be examined as soon as possible to preclude loss of such information. Tampering by the curious and weather are two of the prime contributors to information loss.

The Flight Surgeon should not rely on his memory to place and describe significant findings accurately. He must write a description of what he finds (particularly personal gear) and wherever possible document it with photographs (preferably in color) and laboratory studies as applicable. In the case of fatal accidents he should obtain photographs from various positions and angles to clearly establish the relationship of the body to the wreckage as well as to the immediate environment. The Flight Surgeon should describe to the photographer what he is attempting to record in a particular photograph, so as to enable him to compose the picture so that the area of interest is highlighted, thus adding accuracy to the documentation. Once the wreckage, remains, and personal gear have been moved, photographs may provide the only source of information in establishing a point of vital interest to the investigation. There is one axiom regarding photographs: *Too few can be taken but you cannot take too many.* Once *in situ* photography of the body has been completed and it is removed, the location beneath and adjacent to the remains should be photographed. In removal of bodies an attempt should be made to keep the personal flight gear intact and not to remove it from the body unless necessary to free the remains. Photographs of the cockpit should be made in every possible case—and in several views—showing control settings, instruments and readings, and damaged areas (which later can be related to injuries). The O_2 regulator settings, radio settings, and restraint harness control settings are the most important for the Flight Surgeon.

The wreckage and surrounding area should be carefully searched to locate all body parts, personal equipment, or evidence pertinent to the investigation. A diagram showing the accident distribution pattern must be prepared to clearly identify the location of each item, state the distances between items, and show the direction of the flight path.

Postmortem Procedures

Equipment, Photographs, and X-rays.—When the body arrives at the designated activity, color and black-and-white photographs should be taken of the undisturbed, clothed body (all personal gear still on the body). Past experience indicates that the autopsy suite is the best location for photography and the initial equipment examination. Minimum photography should include anterior, posterior, and right and left lateral views. Closeups of significant areas are made as required in each case. Personal equipment should be removed and carefully examined piece by piece. It is desirable to have a parachute rigger and/or other experienced personnel assist in this examination. Photographs are made of all findings of significance, with special attention to obtaining good, clear detail. The unclothed body must then be photographed in the same manner as described above for the clothed body. Where possible, full-body X-rays are desirable. These films are of value in uncovering unsuspected lesions (particularly skeletal trauma) and foreign bodies. Vertebral fractures (especially cervical fractures) and undetected skull fractures are frequent and should be looked for during X-ray interpretation.

The Necropsy.—The Flight Surgeon *shall* recommend to the commanding officer having custody of the remains that a postmortem examination be performed on *all* persons who sustain fatal injuries in a naval aircraft accident (*Manual of the Medical Department* 17–18(1)). The commanding officer has the authority to sign the permit for autopsy. The autopsy should be performed as rapidly as possible after death to minimize loss of information from degenerative and other changes. The postmortem examination is discussed in the following section on aviation pathology. It is preferable to have the autopsy performed by a military pathologist who has experience in the field of aviation. On occasion, no military pathologist will be available and the services of a civilian pathologist must be obtained. Arrangements in such circumstances can be made through the District Medical Officer. The Flight Surgeon should be present at the autopsy to assist and advise the pathologist. In rare instances, the Flight Surgeon will be required to perform the autopsy himself. An invaluable guide in such cases is NAVMED 5065, *Autopsy Manual.* This publication provides detailed instructions for performing the examination and contains a special section on aviation.

AVIATION PATHOLOGY

Aviation pathology is forensic pathology as applied to aviation. Forensic pathology in general deals with the investigation of suspicious and violent deaths or cases of nonfatal injury and offenses which may subsequently come before the courts. Pathologic anatomy, toxicology, and serology are the basic tools utilized by the forensic pathologist. While aviation pathology does not fit precisely within the definition of forensic pathology, it does utilize the above techniques to solve problems related to aircraft accidents, accident prevention, and research.

The first concerted effort in this field was in the 1930s when the German Air Force began assigning forensic pathologists to accident investigation teams. During World War II in this country, CAPT G. M. Hass of the Army Air Corps did a series of studies of crashes in the Air Training Command and published a number of the early papers in aviation pathology. However, there was little interest in aviation pathology until 1954, when worldwide attention was focused on the *Comet* aircraft accidents. Within a three-month period, two of these commercial airliners crashed into the Mediterranean Sea and salvage of the wreckage was not feasible.

The pathologic findings assumed a role of paramount importance in directing the course of the investigation toward finding the cause of the accidents. These findings indicated that both aircraft broke up in flight and many of the victims free fell into the sea. Studies were undertaken to investigate the structural integrity of the aircraft and eventually it was determined that weaknesses existed in the fuselage which could lead to inflight failure. This deficiency was corrected and no further losses of *Comet* aircraft occurred as a result of fuselage failure.

From these accident studies, aviation pathology gained recognition as a subspecialty in 1954. In March 1955, a symposium on "The Pathologic Correlation of Aircrew Fatalities" was held at the Armed Forces Institute of Pathology (AFIP). Representatives consisted principally of members of the military services of the United Kingdom, Canada, and the United States. From the meeting came the concept of the Joint Committee on Aviation Pathology (JCAP) and on 14 November 1955 the Department of Defense issued a directive to establish the committee with permanent headquarters at AFIP. The current membership includes representatives from organizations coming under the U.S. Department of Defense and from the military services of the United Kingdom and Canada.

The Department of Defense Directive states that:

> The Joint Committee on Aviation Pathology will be concerned with all matters relating to the role of pathology as applied to aviation and flight safety, and will act as a focal point for the dissemination of information on this subject. The specific areas of interest are:
>
> A. Collection of information regarding the correlation between pathological evidence and causative factors of aircraft accidents.
>
> B. Initiation of detailed pathological investigations which may yield information relating to the cause of hitherto unexplained aircraft accidents.
>
> C. Improvement of flight safety records as a result of pathological correlation data.
>
> D. Establishment of a long-range program involving the accumulation of pathological data from a large series of cases.
>
> E. Investigation of psychological and physiological factors which may produce pathological changes as a result of flight stresses.

THE AVIATION PATHOLOGY EXAMINATION

In performing the aviation pathology examination, there are three broad categories of interest:

1. Preexisting disease
2. Environmental factors
3. Traumatic factors

(Each of these categories is discussed and case examples presented in app. 18–4.)

The autopsy should be performed in scrupulous detail. This requirement means a thorough external and internal gross examination, detailed histopathology study, and obtaining tissue specimens for toxicologic and other special studies.

It is beyond the scope of this text to describe in detail every step in the postmortem study. However, some areas of particular importance are discussed below.

In the external examination, particular emphasis should be directed toward the examination of traumatic injuries and burns, and appropriate photographs obtained. Traumatic lesions must be carefully examined for particles of foreign bodies as this can give a clue to their origin.

Specific areas should be examined for various lesions. The head and neck should be examined first. Look for skull fractures which may be identified visually or by palpation. Is there massive fracture of the vault or is there a single area of fracture, possibly with depression? Such clues give an idea of the type and magnitude of the force applied to the skull. If a helmet was on the head when the force application occurred, try to relate areas of damage to the skull injury.

Examine the back of the neck to determine if there is evidence of injury due to rotation of the helmet and impacting of the edge of it against this region. Examine the eyes for subconjunctival hemorrhage and the malar eminences for petechiae. Also look for symmetric round or ovoid contusions on both sides of the mandible just above the chin and for oblong contusions under the chin and around the mouth which are due to oxygen mask and suspension impact. These neck, eye, and face lesions are commonly associated with ejections, particularly those occurring at high speed. The eye and face lesions are seen more frequently than the neck injury in such cases.

Examine the anterior surface of the neck for a linear abrasion lying in a plane between the angles of the mandible and over the larynx. The chinstrap of the helmet often produces such an injury, and where violent impact or high-speed ejection forces are applied to the helmet, it is not uncommon for the abrasion to extend from angle to angle across the neck.

Over the clavicles, essentially at right angles to them, look for linear contusions about 1½ to 2 inches in width. These may be due to being forced against the restraint harness or from the parachute harness.

Examine the region of the upper part of the sternum for a rounded contusion. If the head is violently snapped downward, the chin may contact the chest, resulting in such injury. Occasionally, a serrated pattern of contusion may be seen over the skin of the chest and abdomen which corresponds to the zippers of the flight suit. These lesions generally are indicative of high-magnitude forces which occur in such examples as striking the ground in free fall, low-altitude ejections without parachute opening, and high-impact crashes.

Linear lacerations may be noted which parallel the inguinal ligaments. These are due to restraint and/or parachute harness cutting into these areas. Examine the peritoneum for rupture. High impact forces may produce marked internal pressures which "blow-out" the peritoneum.

Examine the extremities for dislocations of the articulations and fractures. Try to determine if these are due to flailing or indirect trauma. In flail-type injuries, the joint is most frequently dislocated without disturbance of the external surface. On the other hand, direct trauma in many cases will leave a telltale pattern, and/or foreign material from the offending object embedded in the tissues. Lacerations, abrasions, and contusions should be noted and their origin determined insofar as possible.

Burns must be described as to extent and degree. In addition, the source of the burns (i.e., fire, friction, chemical, etc.) should be ascertained.

Two things which are common in high-impact situations are traumatic fat necrosis and pressure tears of the skin. The configuration of the fat necrosis conforms roughly to the outline of the object struck. The epidermis is flattened, smooth, and usually slightly depressed below the surrounding skin surface. Palpation gives a leathery sensation. The color of these lesions varies from reddish brown to an orange-brown hue but the brown character predominates. On sectioning, the underlying fat is semiliquid or completely liquified. Pressure tears of the skin are most frequently seen in the trunk and predominantly are anterior. The skin is laterally pulled apart producing a ragged, torn lesion. The edges of the tear are rough, tend to be slightly everted, and not infrequently there are narrow bridges or bands of tissues which remain intact between the edges.

Certain artifacts are sometimes seen in persons exposed to intense heat. These include fire fractures of the skull, false epidural hematoma, pugilistic attitude, heat amputation of the extremities, and heat evisceration of the abdomen. Fire fractures of the skull occur with greatest frequency in the occipitoparietal area. They have a round appearance, are usually three centimeters or larger in size, and the edges are rough, charred, and friable. It is theorized that steam is generated intracranially and as a result of weakening of the cranial bones by heat, the skull is exploded. False epidural hematomas occur as a result of blood being cooked out of the epidural vessels. This artifact appears as a moderately thin layer of blood extending past the midline, having a bubbly, dark red-black color. Heat produces the pugilistic attitude which describes the flexed position assumed by the extremities. This results from heat-induced contraction of the flexor groups.

Fire amputations appear as spindle-shaped, charred stumps of the extremities. It is not unusual to find the distal end of the bone(s) protruding beyond the remaining charred soft tissue. In heat evisceration, the abdominal wall is markedly charred and thinned and the small bowel blows out through a weakened area as gases expand within it. Absence of other visceral injury is helpful in establishing this diagnosis.

The internal examination should include the head, neck, chest, abdomen, and pelvis in every case. In some cases there may be indication for internal examination of one or more extremities. The following material does not cover the routine examination in the course of an autopsy but is directed toward those aspects of particular interest in aviation.

The internal examination of the head includes several areas of interest. As the scalp is reflected the subgaleal region should be examined for hemorrhage, particularly in the occipital region. Severe head buffet can produce such lesions.

The brain should be examined carefully for evidence of contrecoup injury, tentorial laceration, and herniation of the cerebellar tonsils. Again these lesions can result from high impact forces and head buffet. The dura must be stripped from the base of the skull to examine this region for fractures. Basilar fractures and eggshell fractures (marked fragmentation of the cranial bones) are quite common in aircraft accidents.

Neck structures of particular interest are the hyoid bone and larynx. Fractures of these structures may be overlooked unless they are carefully examined. Hemorrhage, edema, and fractures within the neck are indications of force being applied in this region. The location of hemorrhage and the direction of fracture displacement are helpful in establishing the direction of the force vector.

The pattern of chest injuries varies considerably, being related primarily to the magnitude of the force applied, its relationship to the axes of the thorax, and the surface area of application. The bony thorax should be examined for fractures and dislocations. Point impacts commonly produce injury at the site of force application. On the other hand, compressive forces of high magnitude tend to produce sternal fractures, chondrosternal and costochondral separation, sternoclavicular separation, and rib fractures laterally and posteriorly.

The lungs should be examined for rib markings. These are frequently seen in high-impact and blast injuries. The larynx and trachea should be examined for soot. Layering of soot on the mucosa indicates the person was alive and respiring during exposure to products of combustion. Punctures of the lungs by rib ends or other penetrating objects should be distinguished from ruptures. The same is true of the heart and great vessels. Punctures of the myocardium are generally oval, well circumscribed and sharp edged. On the other hand, ruptures tend to be ragged, linear, and the edges everted. Ruptures of the aorta and pulmonary artery are sharp edged and lie in the circumferential plane of the vessel. These lesions appear as if they had been produced by a sharp instrument and to the uninitiated may be dismissed as artifacts. The three most frequent sites of rupture in the aorta are just above the aortic valve, at the level of the ligamentum arteriosum and at the diaphragm. Ruptures of the cardiovascular system result from a combination of physical displacement and hydraulic forces.

Failure of the diaphragm occurs with a fair degree of frequency. The rupture is most commonly posterior and displacement of abdominal viscera into the thorax is not unusual.

The abdominal and pelvic viscera should be inspected for rupture, internal hemorrhage, interruption of vessels, tearing of attachments, and other evidence of trauma. As in the chest, the type and extent of injury are related to the direction and magnitude of the applied force. In aircraft accidents ruptures of the liver, spleen, kidneys, bladder, and urethra are quite common.

The small bowel may be completely stripped from the mesentery. The stomach is rarely ruptured.

Multiple skeletal fractures occur in many aircraft accidents. Compound fractures, severe comminution, and marked displacement are frequent findings. Fracture dislocations of the vertebral column and pelvis and separation of the symphysis pubica are common findings in high-magnitude force application particularly in the anterior-posterior planes. Decapitation and extremity amputations also are often observed in such circumstances.

In some cases it may be necessary to examine the extremities for foreign bodies or other evidence germane to the investigation. This is determined by the circumstances of each incident.

Toxicology

Routine toxicologic studies should be done in every aircraft accident. These studies are done principally at the Armed Forces Institute of Pathology (AFIP), which is located in Washington, D.C. Although the services of local laboratories should be utilized as indicated for screening purposes, it is strongly recommended that aircraft accident toxicologic tests be referred to AFIP and the results accepted for final reporting purposes.

The tests routinely done at AFIP are lactic acid, carbon monoxide, and alcohol. On special request, studies for drugs or other toxins will be done. However, such studies should only be requested when there is good indication for them. These investigations are time consuming and limit laboratory personnel to one project for a considerable period. Thus good professional judgment is indicated in determining if such procedures are to be requested. It is helpful to indicate to the laboratory what is suspected as a possible toxin when asking for special laboratory tests. If samples of the suspected toxin are available, they should be forwarded to the toxicology laboratory under separate cover and not with the tissue specimens.

Tissues for toxicologic examination must be collected and prepared for shipment with strict precaution to prevent contamination. Formaldehyde, acetone, alcohol, and similar substances must not be allowed to come into close proximity with the tissue specimens. Once remains are embalmed, the possibility of obtaining desired toxicologic studies is nil. For toxicologic studies it is desirable to have at least 250 grams of brain, liver, kidney, and lung, at least 20 cc of blood, and all urine available. Stomach contents should be obtained when indicated. Red bone marrow and lung tissue are useful in cases where there has been severe fragmentation of other viscera. Clotted blood is entirely satisfactory for study.

Individual tissue specimens should be placed in separate plastic bags and heat sealed. Latex rubber bags (condoms) are particularly useful as containers for blood and other liquids. The container should have all excess air expelled before it is sealed (or double knotted in the case of condoms). As a precautionary measure, the specimen should be placed in a second container and an identifying label showing name, rank, service number, date, and place of accident should be securely sealed within this bag. This should be a paper label as plastic labels can result in camphor vapors permeating the specimens and giving false results. Once the individual specimens have been prepared, they should be placed in a deep freeze, dry ice, or other environment in which they will be frozen. When ready for shipment, the specimens should be placed in a heavy cardboard box which is thickly insulated (paper, sawdust, plastic foam, felt, etc.). They should have dry ice packed around the outside of them on all sides and within the center. An easy figure to remember is ten pounds of dry ice for the usual size shipment (i.e., specimens as outlined above). A completed DD–1322 form sealed in a plastic bag must be included in the box. The container should be sealed with tape and then wrapped in several layers of heavy paper and this sealed. Never place dry ice in a sealed container (i.e., thermos bottle, sealed can, etc.) which will not permit the escaping gas to vent or diffuse through its walls. Commercially manufactured dry ice is formed under tremendous pressure; it requires approximately 230,000 cc of carbon dioxide under pressure to form one pound of commercial dry ice. When commercial dry ice is not available, carbon dioxide cylinders (fire extinguishers) can be utilized to produce carbon monoxide snow which may be a satisfactory substitute if a large amount is produced and the carton is well insulated and sealed. The box should be labeled:

The Director
Armed Forces Institute of Pathology
Washington, D.C. 20305
"Rush—Frozen Specimens for Toxicology"

Shipment to AFIP is best accomplished by air freight. Do not send specimens by air express or mail. Once arrangements have been made for shipment, notify AFIP by message (TWX) of the time of arrival, the airline and flight number, and the Washington area airport of arrival. Also request the carrier to have their Washington agent notify AFIP by phone when the shipment arrives.

After the shipment is received at AFIP, the specimens are prepared for the routine studies and any special examinations which may be requested. When these tests are completed and validated, the results are forwarded to the contributor and other appropriate activities.

Lactic Acid.—No attempt is made in this text to develop a detailed account of the metabolic processes leading to lactic acid formation. Only that information of practical application to aircraft accident investigation is presented.

Through experimental studies it has been determined that the only tissue of value in establishing lactic acid levels in aircraft accident victims is that of the central nervous system. Specimens from the cerebral hemispheres are best, with spinal cord being the least desirable. Animal experimentation indicates that increased levels of lactic acid can be demonstrated up to 30 hours after death.

Lactic acid levels of over 200 mg percent in CNS tissue are considered abnormal. Elevations appear to be related primarily to hyperglycemia. At one time it was thought that this test might be specific for the diagnosis of hypoxia which can produce elevated glucose levels. However, it has since been demonstrated that shock, drowning, suffocation, hyperventilation, stress, hanging, and intravenous glucose are some of the things which can result in lactic acid elevations.

Thus the value of lactic acid determinations is principally as an adjunct in establishing a diagnosis. The interpretation of the significance of an elevated lactic acid must be made on the basis of factual information. If there is evidence that an individual was exposed to hypoxia and the lactic acid is elevated, this latter finding adds strength to the diagnosis.

Similarly, if it is thought that an individual was exposed to a stressful situation, where hypoxia was not a factor, the lactic acid may be helpful as a confirmatory finding.

There are certain factors which may lead to erroneous levels of tissue lactic acid. Low levels can result from prolonged exposure of tissue specimens to air, through exposure to water, labor leading to exhaustion, and bacterial contamination. False high levels can be obtained in the case of dehydration and contamination during specimen handling. One other fact must be stressed: the absence of an elevated CNS lactic acid does not absolutely rule out hypoxia.

Although the lactic acid test leaves much to be desired as a specific test, at present it represents the only biochemical tool we have as adjunct in the diagnosis of hypoxia.

Carbon Monoxide.—A blood specimen is preferred for carbon monoxide testing but any tissue which will yield an aqueous extract of hemoglobin can be used. A modified Van Slyke apparatus is used to liberate the carbon monoxide from the carboxyhemoglobin in an aliquot of the extract for analysis. This sample is introduced into the gas chromatograph and the peak height reading obtained. Another portion of the specimen is saturated with carbon monoxide and the peak height determined. The percent carboxyhemoglobin in the specimen is then calculated by comparing the carbon monoxide content with the carbon monoxide capacity.

The carboxyhemoglobin level is a valuable test in determining if an individual was exposed to the products of combustion prior to death. Studies conducted at AFIP indicate that fatalities from aircraft accidents having a carboxyhemoglobin content of ten percent or greater were exposed to carbon monoxide before death.

Although carbon monoxide intoxication per se is a relatively small hazard in modern military aircraft, the detection of carboxyhemoglobin may be of extreme value in an investigation. For example, if lethal injuries are received on impact and there is an elevated carboxyhemoglobin concentration, it means that the individual was exposed to carbon monoxide in flight. Conversely, if there are no lethal injuries, no inflight but a postcrash fire, and the carboxyhemoglobin is elevated, the individual was exposed to products of combustion after the crash.

Therefore, the principal value of carboxyhemoglobin determinations is in establishing whether or not an individual was alive at the time of exposure to the products of combustion. This information is useful in reconstructing the course of events in an accident, particularly from the standpoint of the escape from postcrash fire.

Alcohol.—Statistics available at AFIP indicate that acute alcoholic intoxication is almost nonexistent as a factor in military aircraft accidents. However, in general (private) aviation the converse seems to hold true.

The technique employed at AFIP to determine ethanol levels is based on partition of a volatile compound in solution and the air above the liquid phase. The concentration of alcohol in the air phase is independent of the volume of the solution. Unknown specimens are collected in rubber-capped flasks and analyzed by comparing samples from them with those from standards. Equal volumes of air from over the liquid phase (which is maintained at constant temperature) of the unknown and standard are injected into the gas chromatograph and the peaks recorded. Ethanol quantity can be determined by comparing the unknown peak with that of known standards.

In this country, from the *legal* standpoint it has been fairly widely accepted that persons having 0.05 percent alcohol or less in their blood are presumed to be not influenced and all persons having 0.15 percent or more are presumed to be under the influence. No presumption is made for those showing 0.05 to 0.15 percent. These percentages represent the number of weight units of alcohol per 100 weight units of specimen.

These levels, it should be noted, are based on *legal* considerations. There is extensive data indicating that psychomotor skills are affected by concentrations of less than 0.05 percent blood alcohol levels. The relationship of diminished psychomotor capability to aviation skills requires no amplification.

Drugs.—When drugs are found in the individual's personal possessions or, in the course of the investigation, it is determined that the deceased was taking medication, a tissue analysis must be given strong consideration. This is particularly true when there is no evident cause of an accident. The most valuable tissues for drug analysis are brain, liver, kidney, and blood. In some cases other specimens can be used but as general rule they are unsatisfactory. Urine can be used for detecting certain drugs which are excreted through the kidneys. As noted earlier, the toxicologist should be informed of the drug or class of drug suspected.

Miscellaneous Toxins.—Hydraulic fluids, fire extinguishants and thermal decomposition products of lubricants, plastics, and waxes are just a few of the toxins which may occur in the aviation environment. When there is suspicion of an unusual toxin, it is highly advisable to seek the advice of an experienced toxicologist in conducting the investigation.

Drowning.—The best diagnostic tool to establish drowning is the finding of diatoms in the kidney, brain, and liver. These are unicellular skeletons of plankton whose walls are impregnated with silica. They occur in most bodies of water throughout the world. When water containing diatoms is drawn into the lungs, these structures may pass through the pulmonary vascular bed into the greater circulation and ultimately lodge in organs. Finding diatoms in the lungs alone is not considered sufficient evidence to make a positive diagnosis of drowning. In very short-duration submersion, this finding might be considered presumptive evidence of drowning but experimental work has shown that with prolonged submersion diatoms can be washed into the lungs.

The Gettler test is based on finding a difference in sodium chloride levels in the right and left heart. Normally, sodium chloride levels are the same in both sides of the heart. However, if salt water is inhaled during drowning, it may be carried to the left side of the heart thus giving a higher sodium chloride content on the left than right. The converse is true if fresh water is inhaled due to dilution occurring on the left side. This test is much more reliable for drownings occurring in salt water than those occurring in fresh water.

Bloodstains and Serology

The identification of stains and smears is a common problem in accident investigations. The vast majority of such incidents involve the establishment of whether or not a suspicious area represents bloodstaining. A simple way of determining the answer is to apply the dampened end of a bloodtest strip (Hemastix, Ames Laboratory, FSN 6505-890-1549) for 15 to 20 seconds to the suspected area. A positive reaction will occur if blood is present. This technique has been utilized by accident investigators for several years and experience indicates that it is very reliable.

Blood typing is indicated as part of the investigation in some cases. In multiplace aircraft this may be a useful procedure in establishing the position of various crewmembers at the time of the accident when other methods of identification are unavailable. It is also of value occasionally in determining who was struck by a part of an aircraft structure when there is evidence of such an occurrence. Inability to obtain a satisfactory specimen for typing and persons having the same blood type are two of the most frequently encountered limitations to the value of such tests.

The precipitin test is valuable in determining whether blood or other tissue specimens are of human or animal origin. In accidents due to bird strikes, this can be a most useful test to provide confirmatory evidence in the investigation.

Various Federal, State, and local laboratories are capable of performing serologic tests. The availability of such services should be ascertained for a particular locality and a protocol obtained from the laboratory which describes the desired method for handling specimens.

Massive Injury

It is not unusual in high-impact situations to find massive injury. Decapitation, amputation of extremities, avulsion of the abdominal wall and thorax with loss of contents, and complete body fragmentation are examples of what may occur. In the case of fire, marked incineration of the body is possible. When there is loss of tissue, it becomes obvious that the postmortem examination must be limited in scope. However, every effort should be made to accomplish all possible forms of examination. There are a number of cases on file at AFIP in which massive injury occurred but by meticulous and persistent efforts significant aeromedical factors were demonstrated.

Identification

Identification, as a rule, poses no problem except where there is massive injury. In such instances dentition, one or more printable finger pads, peculiar anatomic malformations, characteristic scars, and unusual tattoos are valuable in establishing positive identification. Blood type, items of wearing apparel, personal effects, and identification tags are useful only to the extent of making a presumptive identification. Local law enforcement agencies can often provide assistance in establishing identification. Whenever there is doubt about identification, it is wise to request the services of an individual who is an expert in this field. This may avoid complicated legal problems which often arise when there is confusion in identification. Ordinarily there is no problem involved with military pilots and aircrew lost at sea, for precedents have been established in declaring such individuals missing and presumed dead.

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APPENDIX 18-1

INTERVIEW PROCEDURES

IF THE Flight Surgeon is to obtain usable information from eyewitness accounts, he must understand the proper principles of interviewing. Warren (1954) lists the rules for interview procedure found in the *International Civil Aviation Organization Manual of Aircraft Accident Investigation*. These are:

1. It is not as a rule difficult to get witnesses to tell what they saw if the purpose to be served is made clear to them. The investigator should assure the witness that the main purpose for collecting all possible factual evidence concerning the accident in question is in order to prevent similar accidents in the future and to safeguard lives.
2. It should be emphasized to the witness that he should speak only of matters within his personal knowledge; that is to say, what he himself saw and heard, and not hearsay evidence.
3. The witness should begin by giving his name, address, occupation, and aviation experience, if any, and, if a child, his age.
4. He should be encouraged to tell in his own words all that he personally knows about the accident.
5. While he is giving his story, he should not be interrupted, except to prevent him from going into irrelevant matters.
6. After he has finished, questions should be put to him to clear up any doubtful points which may arise on his statement, but questions should *not* be phrased in such a manner as to suggest answers.
7. The evidence which he gives should be taken down in writing in his presence. It may be taken down by a stenotypist to save time, or it may be taken down by the investigator himself, or, except where it would conflict with local procedure, the witness may hand in a written statement to the investigator.
8. When the evidence of the witness has been completed, he should sign the record which has been taken of it.
9. The use of highly technical terms in putting questions to a witness who may have no knowledge of aeronautics should be avoided.
10. A witness should be treated with the utmost courtesy at all times, and any semblance of coercion avoided.
11. Some people object to being involved in inquiries about accidents, and the witness should be reassured as to the sincerity of the purpose of the investigation, and his duty

as a member of the public to tell what he knows, in the general interest, should be impressed upon him.

12. A witness may be able to express a statement better by sketches than by words. Such sketches are acceptable as clarification of his evidence and should be signed by the witness as a part of his statement.

THE INTERPRETATION OF VERBAL TESTIMONY

Having obtained the testimony of eyewitnesses or survivors, the Flight Surgeon must make an evaluation of this evidence. The rule concerning the interpretation of such evidence is that it should be evaluated in the light of other facts known concerning the crash. Unfortunately, however, the verbal testimony may be all that is known about the accident.

Many instructors in introductory psychology use a time-honored device to illustrate graphically the way in which eyewitnesses may go astray in their accounts of events. The instructor and one or two cohorts will stage a small altercation at one side of the classroom, with each participant acting completely in accordance with a prearranged script. Every member of the class will feel that he has observed exactly what took place, however surprised he might have been to see it happen. When the written accounts of the incident are collected and examined, however, it is usually found that they range from the reasonably accurate to completely outlandish accounts submitted in good faith by students who might just as well have been sitting in another classroom. Such a demonstration is not provided in an attempt to deprecate eyewitness accounts, but rather to instill a spirit of caution in those called upon to interpret them.

In the case of an aviation crash, events occur with extreme rapidity. This is, unfortunately, the condition least conducive to reliable accounting. For this reason, the Flight Surgeon should attempt to follow these rules in evaluating evidence obtained from eyewitnesses:

1. If reports can be obtained from two or more independent observers of the event, considerable reliance can be placed upon those portions of the reports which are in concurrence.

2. Errors of forgetting occur rapidly. Warren (1954) estimates that we ordinarily forget 50 percent of what we learn within 24 hours. For this reason, the sooner after the accident the testimony of witnesses can be obtained, the more reliable it is likely to be.

3. If the survivor of an accident is showing physiological signs of a shock condition, the accuracy of his testimony should be suspect. In some accidents, the results of injury or shock may include a loss of memory for the events of the accident and even events before and after the accident (Warren, 1954).

4. Testimony should be evaluated in terms of realities of the situation. For example, if the circumstances of the crash indicate the pilot may have been guilty of some infraction of good safety practices, the Flight Surgeon should expect his fellow pilots who

may have been witnesses to follow a normal course of action and attempt to protect their friend in their discussion of the accident.

In summary, it can be said that eyewitness accounts can prove extremely valuable in pinpointing the causes of accidents or in suggesting additional avenues of investigation. Wherever possible, however, eyewitness accounts should be evaluated in the light of additional evidence from other sources which can be brought to bear upon the investigation.

APPENDIX 18–2

GENERAL DEATH PROCEDURE CHECKLIST

TO FACILITATE and more expeditiously handle the administrative details encountered at the time of a fatal accident, it is recommended that the following checkoff list, along with an adequate number of ready packets of all forms and letters, be prepared and kept available to personnel concerned with handling of deaths.

1. Log all information in MOOD's log.
2. Identify remains and tag body with DD Form 1380 (U.S. Field Medical Card).
3. Notify the following by telephone, first obtaining name and rank of person answering telephone.
   a. Medical Officer of base.
   b. Medical Administrative Officer (where one is assigned).
   c. Commanding officer or duty officer of activity to which deceased is attached. If deceased's command cannot be reached by telephone or messenger, notify by dispatch.
   d. Duty officer of base.
   e. Local authorities. Notify medical examiner, coroner, or other responsible local official of death and obtain permission to move remains. Prior agreement facilitates this action.
   f. Senior chaplain (state religion if known).
   g. Alert OOD at Naval Hospital where remains will be sent or undertaker under contract where no Naval Hospital is available.
   h. Alert pathologist and arrange for postmortem examination. Supervise preparation of specimens and insure proper shipping.
4. Clerical procedures to be accomplished:
   a. Inventory of receipt of personal effects on the remains. Personal effects are delivered to deceased's commanding officer and same is receipted for by commanding officer or his OOD (local form).
   b. Letter of release of remains to Naval Hospital or contracting undertaker (local form).
   c. Letter of receipt of remains from Naval Hospital or contracting undertaker (local form).
   d. Letter of request and authorization for autopsy with Standard Form 523 (Authorization for Postmortm Examination) as an enclosure. Autopsy shall be reported

on Standard Form 503 (Autopsy Protocol) and DD 1322A. Request copies of Standard Form 503 to attach NAVMED Form N which is forwarded to BUMED, MMN 17–18 (3), and to enclose with Medical Officer's Report of Aircraft Accidents, Incidents, and Ground Accidents, OPNAV Form 3750–8 (Rev. 3–63).

e. Insure preparation of NAVMED 609 (Report of Disposition and Expenditures Remains of Dead). (MMD 17–9B).

f. Insure preparation of NAVMED Form N (Certificate of Death). (MMD 17–10, 17–11, 17–4).

g. Insure preparation of State Death Certificate as necessary.

h. Insure preparation of Medical Officer's Report of Aircraft Accidents, Incidents and Ground Accidents, OPNAV Form 3750–8 (Rev. 3–63).

i. Admit to sick list and dispose of as DD.

j. Include in monthly report NAVMED 1454B (BUMEDINST 6310.6 series).

k. Close out Health Record and mail (MMD 16–9, 16–12).

APPENDIX 18-3

EMERGENCY SUPPLIES AND EQUIPMENT

EMERGENCY supply and equipment requirements are determined by the type, size, and location of the activity. As an example, the Emergency Material Instruction of the Medical Department of a Marine Corps Air Station is printed below. This illustrates the requirement of a particular activity and is presented as a guideline to the reader.

1. *Purpose.* To promulgate a list of materials to be carried in the Medical Department's Crash Bags, Flight Surgeon's Bag, Specimen Collecting Kits, and Emergency Vehicles in order to insure that they are constantly in a state of readiness.

2. *Background.* In order for the Medical Department to meet its assigned mission, it is necessary to maintain all emergency equipment in a constant state of readiness. Therefore, a list of materials has been compiled to enable medical personnel to cope with the majority of emergency medical situations encountered.

3. *Instructions*

a. It shall be the responsibility of the Senior Corpsman assigned to the Emergency Dressing Room to maintain the emergency equipment in a constant state of readiness.

b. He shall inventory the emergency equipment on the first working day of the month and immediately correct any discrepancies. Every effort should be made to inventory the vehicles in a manner that will not impede the emergency use of same during the time of inventory. Whenever the Crash Bag seal is broken an inventory must be completed immediately.

c. The inventory log will be signed by the corpsman. Any major discrepancies will be reported in writing via interoffice memo to the Crash Officer.

4. *Location*

a. *Crash Bags.* They will be located in the Duty Crash Wagon and the Metropolitan Ambulance during the hours of 0630 to sunset or secure from flight quarters, whichever is later. They will then be kept in the Corpsmen's Duty Room under the cognizance of the Chief of the Day. This precaution will be taken to avoid the possibility of theft of the narcotics contained in the Crash Bags.

b. *Flight Surgeon's Bag.* It will be kept in the Duty Crash Wagon at all times.

c. *Specimen Collecting Kits.* There will be six kits maintained in the following locations:

(1) Metropolitan Ambulances—one (1).
(2) Duty Crash Wagon—one (1). (Placed in the Flight Surgeon's Bag.)
(3) "Off Duty" Crash Wagon—one (1).
(4) Record Office—three (3). (Placed in the file cabinet with the Medical Officer's Aircraft Accident Reports (MOR) Forms. These are to be checked out to Medical Department personnel accompanying squadrons on deployment.)

INVENTORY OF CRASH BAGS

Drugs

1.	Aminophylline injections, 7-½ gr	2 vials
2.	Ammonia inhal	6 amps
3.	Alcohol, 70 percent	2 ounces
4.	Bacitracin ointment	1 tube
5.	Brandy, 2 ounces	2 bottles
6.	Caffeine—sodium benzoate	1 amp
7.	Epinephrine, $\frac{1}{1000}$	1 vial
8.	Lidocaine, 1 percent	1 vial
9.	Merthiolate tincture	2 ounces
10.	Morphine syrettes	5
11.	Soap phisohex	2 ounces
12.	Sodium Amytal	1 unit
13.	Atrophine injection syrettes	1
14.	Nembutal, 100 milligrams	6 caps
15.	Wyamine	1 bottle (10 cc)

Ophthalmic ointments

1.	Sulfacetamide	1 each
2.	Tetracaine eye drops	1 each

Dressings

Bandaids	20
Ace bandages, 2-inch	2
Ace bandages, 3-inch	2
Ace bandages, 6-inch	1
Eye pads	4
Gauze squares, 2 x 2 inches	1 package
Gauze squares, 4 x 4 inches	1 package
Vaseline gauze	4 packages
Roller gauze, 1-inch	1 roll

Dressings—Continued

Roller gauze, 3-inch	2 rolls
Tape, 2-inch	1 roll
Triangular bandage	2
Battle dressing, small	2
Battle dressing, medium	2

Sterile supplies

1. Cotton swabs in tube	15
2. 18-gauge needle	2
3. Knife blades in solution 10, 11, and 15	1 each
4. 18-gauge needle as tracheotome	1
5. Suture set with 4–0 silk and 4–0 gut with needles, knife, and blades	1
6. Assorted hypo needles, 26, 23, and 2-gauge	2 each
7. Syringe, 2 cc	1
8. Syringe, 5 cc	1
9. Syringe, 10 or 20 cc	1
10. Sterile towels	4
11. Tracheotomy set	1

Other supplies

1. Blood pressure cuff	1
2. Vial files	2
3. Flashlight with batteries	1
4. Pencil, skin marking	1
5. Bandage scissors	1 pair
6. Splint, mesh roll	1
7. Stethoscope	1
8. Tongue depressors	15
9. Tourniquet	1
10. Resusitube	1
11. Emergency medical tags	1 book

Inventory of Flight Surgeon's Bag

1. Rubber body bag	1 each
2. Flight suit	1 each
3. APH–5 helmet	1 each
4. Specimen collecting kit (packed in a manner that will prevent breakage). *The following items will be boxed together to prevent breakage:*	
5. IV set	1 set
6. Adhesive plaster, 1-inch	1 roll
7. Arm board	1 each
8. Tourniquet	1 each
9. Dextran, 500 cc	1 bottle

Inventory of specimen collecting kit

1.	Copy of Memo from Medical Officer, MCAS, to Pathology Dept., Naval Hospital containing instructions for collecting blood and tissue specimens.	
2.	5-milligram vacutainer tube, oxalate, marked "Glucose"	2
3.	10-milligram vacutainer tube, oxalate, marked "Alcohol"	2
4.	10-milligram screw-top tube, oxalate, marked "C.O."	2
5.	Paraffin block	1
6.	Mineral oil, 4-ounce bottle	1
7.	Syringes, 50, 10, and 5 milligrams	1 each
8.	Wide-mouth jar, oxalate, marked "Add 50 ml blood"	1
9.	Vacutainer adapter	1
10.	Knife handle	1
11.	Blade, surgical, 20G, size 2, Type AA	1 package (6)
12.	Needles, in sterile tube, 13-, 20-, and 21-gram vacutainer	2 each
13.	Plastic specimen bags—4 sizes	10
14.	Matches, safety	1 box
15.	Gloves, surgical, size 8	2 pairs
16.	Gauze squares, 4 x 4 inches, used in packing	
17.	Copy of "Direction for Shipping Tissue Specimen"	1

INVENTORY OF AMBULANCES

Metropolitan Ambulance

1. Wheeled ambulance stretcher
2. Resuscitator
3. Spare oxygen wrapped in towel
4. Straightjacket
5. Restraint set with key
6. Blanket
7. Splint set
8. IV set (2)
9. IV solutions (Dextran, 5% G/W, N. Saline)
10. Crash bag
11. Specimen collecting kit
12. Sheets, sterile (4)

Duty Crash Wagon

1. Resuscitator
2. Stokes stretcher
3. Army litter
4. Oxygen
5. Bags
6. Splints
7. Foul weather gear
8. Sheets
9. Crash Bag
10. Flight Surgeon's Bag
11. IV sets (2)
12. IV solutions (Dextran, 5% G/W, N. Saline)
13. Sheets, sterile (4)

INVENTORY OF AMBULANCES

Off-Duty Crash Wagon

1. Resuscitator
2. Army litters (2)
3. Unit One
4. Poncho
5. IV sets (2)
6. IV solutions (Dextran, 5% G/W, N. Saline)
7. Specimen Collecting Kit
8. Sheets, sterile (4)

It is recommended that the following items be included in the Medical Investigator's kit:

1. 35mm camera with color film
2. Heavy rubber or leather gloves
3. Self-adhesive labels
4. Heavy cardboard tags with tie string
5. China-marking pencil
6. Indelible pencil
7. Hemastix
8. Small vial of physiologic saline
9. Dental inspection mirror
10. Scalpel, thumb forceps, and small hemostats
11. Plastic bags, assorted sizes (with ties)
12. Plastic pill bottles, assorted sizes
13. Clipboard with grid paper
14. Magnifying glass

APPENDIX 18-4

AVIATION PATHOLOGY CATEGORIES AND CASE HISTORIES

PREEXISTING DISEASE

This category includes all organic disease processes, known or unknown, which existed prior to an accident. The most important aspect of such cases is establishing whether or not the process represented a causative or contributory factor in the accident.

Statistics compiled at AFIP show that coronary artery atherosclerosis is the most common preexisting disease process of significance found in aviation personnel. Mason, Townsend, and Jackson (1963) reported a series of 13 cases known to JCAP in which coronary artery disease was implicated in accidents or incidents. After preparation of their paper, seven additional cases were uncovered.

Following are two case histories which illustrate an incident and an accident from coronary artery disease:

CASE ONE: A 34-year-old copilot of a C-46 remarked that he felt ill on an instrument landing approach. Shortly thereafter he clenched his fists, flexed his elbows, stiffened in his seat, and lifted his arms above his shoulders in a rigid position. He then slumped forward and began gasping for breath. Pulse was rapid and irregular and following landing the copilot was pronounced dead. Autopsy revealed moderately advanced atherosclerosis of all coronary arteries. There was no evidence of infarction. It was assumed that he suffered an acute coronary insufficiency, without thrombotic occlusion, resulting in an arrhythmia which produced his death.

CASE TWO: A 40-year-old Naval Aviator was making bombing and strafing runs on surface ships. On the final run, the aircraft nosed up, returned to a horizontal attitude and then dove into the side of a ship. Examination of the aircraft wreckage showed no evidence of failure of any part of the plane.

Autopsy showed marked atherosclerosis of the anterior descending branch of the left coronary artery with acute thrombotic occlusion. The myocardium revealed scattered foci of fibrosis.

Other preexisting disease processes which have been discovered include colloid cyst of the third ventricle of the brain (three cases), renal cell carcinoma, rhabdomyosarcoma, hypoplasia of the kidney, glomerulonephritis, bronchiectasis, and goiter. This by no means represents the entire spectrum of processes which have been discovered but does provide some idea of what may exist in a relatively young population.

ENVIRONMENTAL FACTORS

Areas of interest in environmental factors include:

(1) Altitude
(2) Speed
(3) G forces
(4) Toxins (including alcohol and drugs)
(5) Temperature
(6) Noise and vibration
(7) Stress

All of these factors represent parts of the hostile aviation environment and their potentially harmful effects are well established physiologically and psychologically. The primary interest of the Aviation Pathologist is in establishing the relationship of these factors to the cause of accidents and the production of injuries and death.

Following are several case histories which illustrate the effects of some environmental factors:

CASE ONE: A transport-type aircraft made a controlled crash landing in a wooded area. Deceleration forces were relatively low and there was little structural damage to the aircraft. An intense fire developed after initial impact which eventually burned out the entire fuselage. The pilot and flight engineer escaped but 74 individuals died. Postmortem examination failed to reveal a single lethal injury in any of the victims. However, all showed elevated carboxyhemoglobin levels indicating death from carbon monoxide intoxication. The distribution of the bodies of the victims indicated that an attempt was made to escape from the rear cabin door which apparently was distorted and could not be opened. The need for additional escape routes was established by this investigation.

CASE TWO: A 33-year-old pilot was flying at a cabin altitude of 22,000 feet in a T-33. He complained to the observer in the rear cockpit of having chest pains after 40 minutes of flight and elected to return to the home base. The observer took control of the aircraft and within five minutes the pilot was unconscious. The observer landed the aircraft and when medical personnel arrived they found the pilot unconscious and in shock. He remained in shock until death occurred approximately 12 hours later. Autopsy showed an obese male with abdominal subcutaneous fat measuring up to five centimeters. The liver was fatty, the heart enlarged, a patent foramen ovale present, and the lungs and brain edematous. Microscopic sections revealed fat emboli in the lungs, brain, and kidneys. These findings lead to a diagnosis of decompression sickness as the cause of death.

Case Three: An observer ejected from the rear cockpit of a high-performance jet aircraft at low altitude. Speed at time of ejection was determined to be over 500 knots. The observer was found dead 12 hours after the accident. Evidence indicated that he had survived for several hours. Autopsy findings included marked traumatic emphysema, focal atelectasis, and hemorrhage in the lungs. This pattern was interpreted as a "blast" type injury to the lungs as a result of exposure to the high forces encountered on entering the airstream during the ejection sequence.

TRAUMATIC INJURIES

Traumatic injuries are fairly common in aircraft accidents. The primary concern of the Aviation Pathologist is determining the source of these injuries and their prevention through modification or removal of the causative factor.

The following cases illustrate the effect of traumatic factors:

Case One: A pilot ejected from a jet aircraft after it became uncontrollable. Windblast rotated the protective helmet upward and backward resulting in the edge of the helmet being pressed against the back of the neck. Head buffet also occurred as a result of the windblast, and the posterior cervical area was struck several sharp blows by the edge of the helmet. This resulted in a fracture dislocation of C5 with transsection of the spinal cord.

Case Two: The copilot of a transport-type aircraft was killed when a crash occurred on takeoff. Autopsy showed a massive, slashing-type injury which was attributed to being hit by a propeller. Further investigation showed failure of the restraint harness, which allowed the individual to be thrown from the aircraft into the path of the prop.

Case Three: A pilot made a controlled ejection from a jet fighter after the engine failed, and it was known that the parachute had deployed normally. He was found dead several hours later. Examination revealed severe head and back injuries which produced death. Subsequent study indicated that the pilot had been struck by the ejection seat after he had separated from it and the fatal injuries were sustained at that time.

CHAPTER 19

Casualty Management and Aeromedical Evacuation

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Naval Aerospace Medical Institute

General
Acquisition Phase
  Aircraft Carrier
  Other Vessel
Triage Phase
Emergency Treatment Phase
  Air Transportability
  Evacuation Decision
  Communications with MAC
  Emergency Treatment and the Walking Blood Bank
Initial Transport Phase
  Patient Preparation
  Transport from Sickbay and Aircraft Loading
  Emergency Patient Care En Route
  Holding Facilities at Transfer Point
  Patient, Records, and Property Transfer
Final Transport Phase
References

GENERAL

PATIENT ACQUISITION, transportation, and early care represent important determinants of morbidity and mortality. The armed forces have recognized the desirability of early acquisition and care for many years, and continuous development of new techniques and equipment has caused a steady decrease in acquisition time (i.e. time until initial hospital treatment).

The Navy's casualty evacuation problems afloat have changed somewhat recently, as have those of the Marine Corps ashore. Helicopter evacuation of casualties from smaller vessels in the train to well-equipped capital ships has lessened some risks and increased others, but has certainly speeded up transportation. The same is true of helicopter evacuation of wounded directly from the battlefield to a hospital or hospital ship. Casualties can now be evacuated from southeast Asia to the continental United States via the Aeromedical Evacuation (AME) flights of the Military Airlift Command (MAC) in less than 24 hours. During these flights, patients have the benefit of a competent and well-trained staff, with all necessary equipment aboard the aircraft to care for their needs. Technical, administrative, and medical problems have been thoroughly worked out.

The adoption by MAC of high-performance jet transport aircraft for AME has allowed transport of men who might not have been permitted AME ten years ago. The C–141 *StarLifter,* shown in figure 19–1, is a long-range, high-speed jet aircraft designed especially for the Air Force logistics supply system. It was introduced into the Military Airlift Command in 1965. It cruises at slightly over 500 mph and can deliver patients

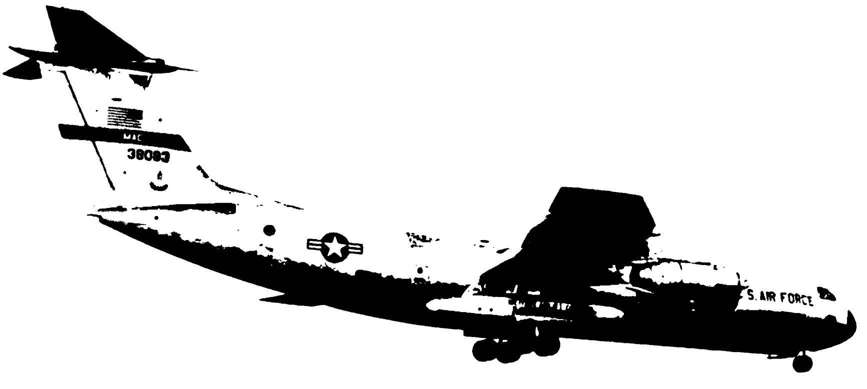

FIGURE 19–1. *C–141* StarLifter, *MAC primary transoceanic casualty evacuation aircraft* (USAF PHOTO).

from Japan to Travis AFB in just nine hours. In flight, the aircraft can be pressurized for an 8,000-foot cabin altitude at 40,000 feet or for sea level pressure up to 21,000 feet. The higher operating altitude of the *StarLifter* as opposed to earlier piston craft, avoids most turbulence and results in better patient tolerance of the evacuation.

As shown in figure 19–2, there is a minimum exposure of the wounded to the weather as they are transferred directly from an ambulance parked under the cargo doors, up the rear loading ramp, and into the aircraft. Figure 19–3 shows the interior of the aircraft, with patients ready for evacuation and with the built-in oxygen equipment ready for immediate use as required.

The primary aircraft used by MAC for evacuation flights within the United States is the C–131 *Samaritan*, shown in figure 19–4. This is a low-flying

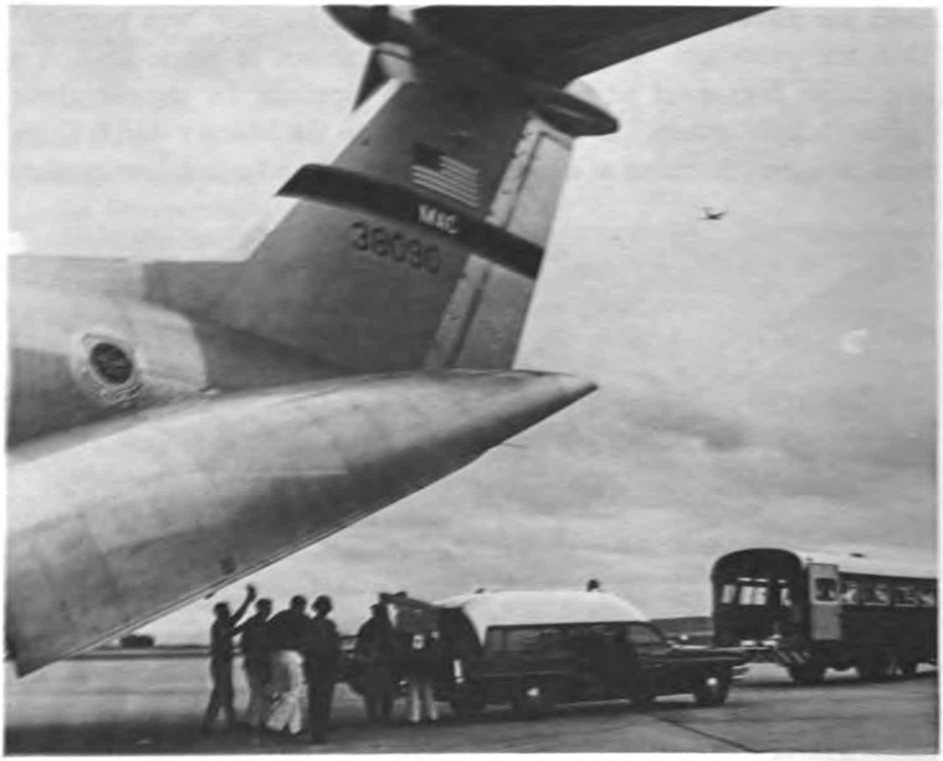

FIGURE 19–2. *Transfer of casualty from Vietnam ambulance into C–141 StarLifter* (LOCKHEED-GEORGIA COMPANY PHOTO).

plane capable of carrying twelve litter and 28 ambulatory patients. It is configured with special medical equipment to handle all types of cases, ranging from polio victims in iron lungs to babies in incubators.

It is the policy of the Department of Defense that in both peace and war the movement of patients of the armed forces shall be accomplished by airlift when available and when conditions are suitable for aeromedical evacuation, unless medically contraindicated.

OPNAVINST 4630.9 series defines several types of AME, including strategic, tactical, and forward. The Military Airlift Command is assigned responsibility for all *strategic* AME, except that the Navy is responsible for AME within naval (including Marine) combat areas and over routes of sole interest to the Navy where the facilities of the Air Force, including MAC, cannot provide the service. The Air Force component commander is responsible for all *tactical* AME for the U.S. armed forces except as noted above. *Forward* AME is that portion of AME "which provides airlift for patients

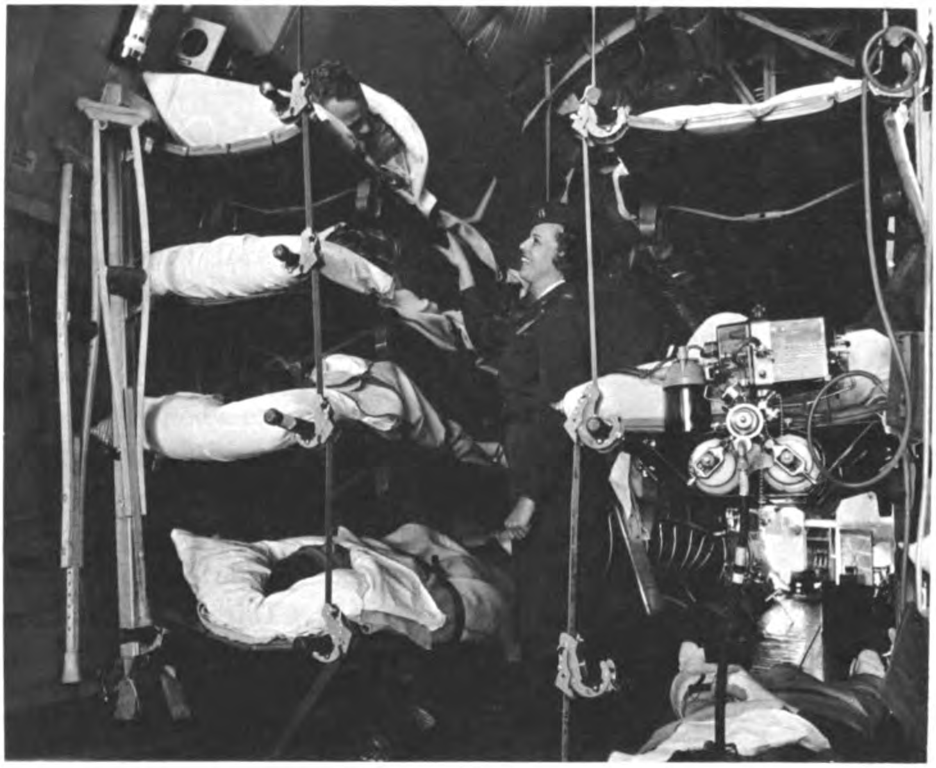

FIGURE 19-3. *Interior of C-141* StarLifter (LOCKHEED-GEORGIA COMPANY PHOTO).

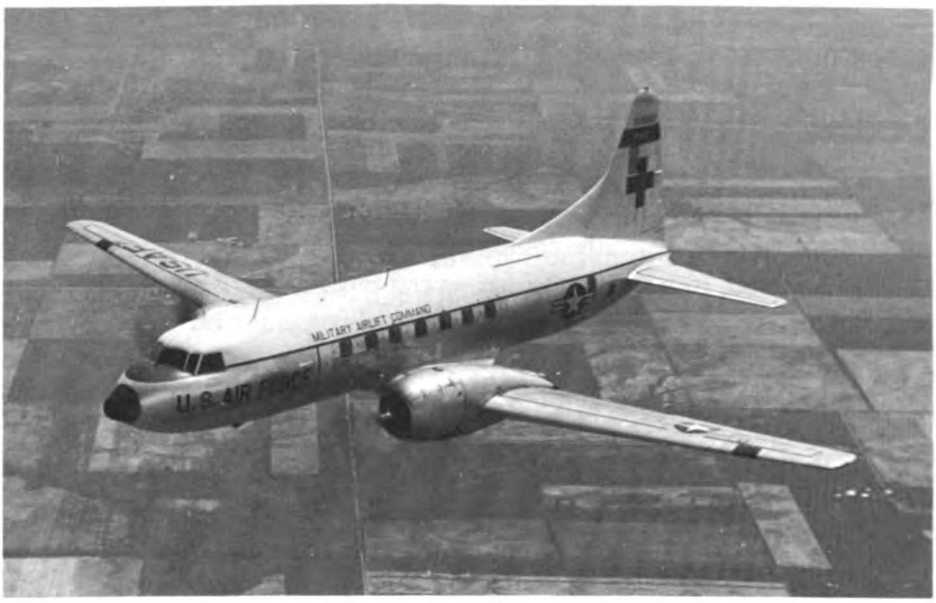

FIGURE 19–4. *C–131A* Samaritan, *MAC primary aircraft for evacuation flights within the United States* (USAF PHOTO).

between points within the battlefield, from the battlefield to the initial point of treatment, and to subsequent points of treatment within the combat zone."

Within the limits of these definitions, the types of AME with which the Navy Flight Surgeon is concerned are the forward and tactical types. However, these terms have little relevance to the aircraft carrier at sea, and the problem of the Navy Flight Surgeon outside the United States is in acquisition of the casualty, early treatment, and evacuation to a hospital, hospital ship, or MAC AME flight, for those patients who require definitive care not available aboard.

For purposes of clarity, the acquisition and handling of casualties on aircraft carriers and smaller vessels at sea have been divided into five phases in a somewhat arbitrary manner (see figure 19–5). This allows an orderly discussion of these matters, despite the fact that management of casualties at sea is rarely so orderly. Such divisions do, however, allow a better understanding of needed steps and decision points. Although this discussion is limited to vessels at sea, there is some carryover for vessels docked in foreign ports.

ACQUISITION PHASE

Aircraft Carrier	*Other Vessel*
Emergency First Aid	Emergency Treatment
Selection of Stretchers	Radio Consultation
Selection of Route	Selection of Transfer Method
Prior Training and Inspection	Highline
	Stretcher
	Bosun's Chair
	Helicopter
	Small Boat

TRIAGE PHASE
- Establish Working Diagnosis
- Determine Priorities for Care

EMERGENCY TREATMENT PHASE
- Air Transportability
- Evacuation Decision
- Communications with MAC
- Emergency Treatment

INITIAL TRANSPORT PHASE
- Patient Preparation
- Transport from Sickbay and Aircraft Loading
- Emergency Patient Care En Route
- Holding Facilities at Transfer Point
- Patient, Records, and Property Transfer

FINAL TRANSPORT PHASE
- MAC Aeromedical Evacuation Flight

FIGURE 19–5. *Phases in acquisition and handling of casualties at sea.*

ACQUISITION PHASE

Aircraft Carrier

When a casualty occurs, shipmates should give immediate first aid to the victim and notify the medical department. However, first aid is not always rendered because of fear of making the injury or illness worse. Confusion is usually present, and there sometimes is delay in informing the medical department. In many cases, notification to sickbay consists only of a request for a corpsman, with no information furnished as to the injury or illness. Location of the casualty is sometimes given incorrectly, as, for example, "near the barbershop," when there are three barbershops on board, rather than correctly, giving frame number, section, and compartment number. Only when a member of the medical department arrives, in the usual case, does the patient begin to receive first aid. (Note: the Flight Surgeon should *never* put a red lens in his emergency flashlight, because such light does not reveal blood, and bleeding is one of the first signs he must look for.) The hospital corpsman may have arrived before the medical officer, in which case proper first aid will already be underway. Corpsmen bring a first aid bag and a resuscitator to every emergency call on the ship, so the medical officer can be certain that these items of equipment will be available at the scene.

If there is a disaster such as fire or explosion, the corpsman and medical officer must stay clear of repair parties fighting the fire or other damage and limit their efforts to care of the sick and wounded. The repair party is trained and equipped to perform rescue operations, and such operations are not within the purview of the medical department unless directed by competent authority. In a disaster in which a number of repair party personnel are injured or killed, all members of the medical department, officer and enlisted, may be required to perform rescue operations in addition to caring for the injured. Every possible aid must be given to prevent hemorrhage, suffocation, or further increase in injury during the rescue process.

When first aid has been given, the casualty must be transported rapidly to sickbay in such a way as to minimize the possibility of further injury or aggravation of existing injury. To achieve these goals, the medical officer or hospital corpsman may select stretcher type and evacuation route to sickbay.

Three types of stretcher are available aboard ship:

1. Stokes.
2. Semirigid (Neil-Robertson).
3. Field (pole litter).

The Stokes stretcher is a wire basket with a tubular frame, contoured to give support to the occupant and to keep the frame between the patient and possible impacting objects. It has a wooden slat frame in the torso section, lines attached at head and foot for lifting, and straps at torso and midleg to keep the patient within the stretcher despite considerable movement. It has

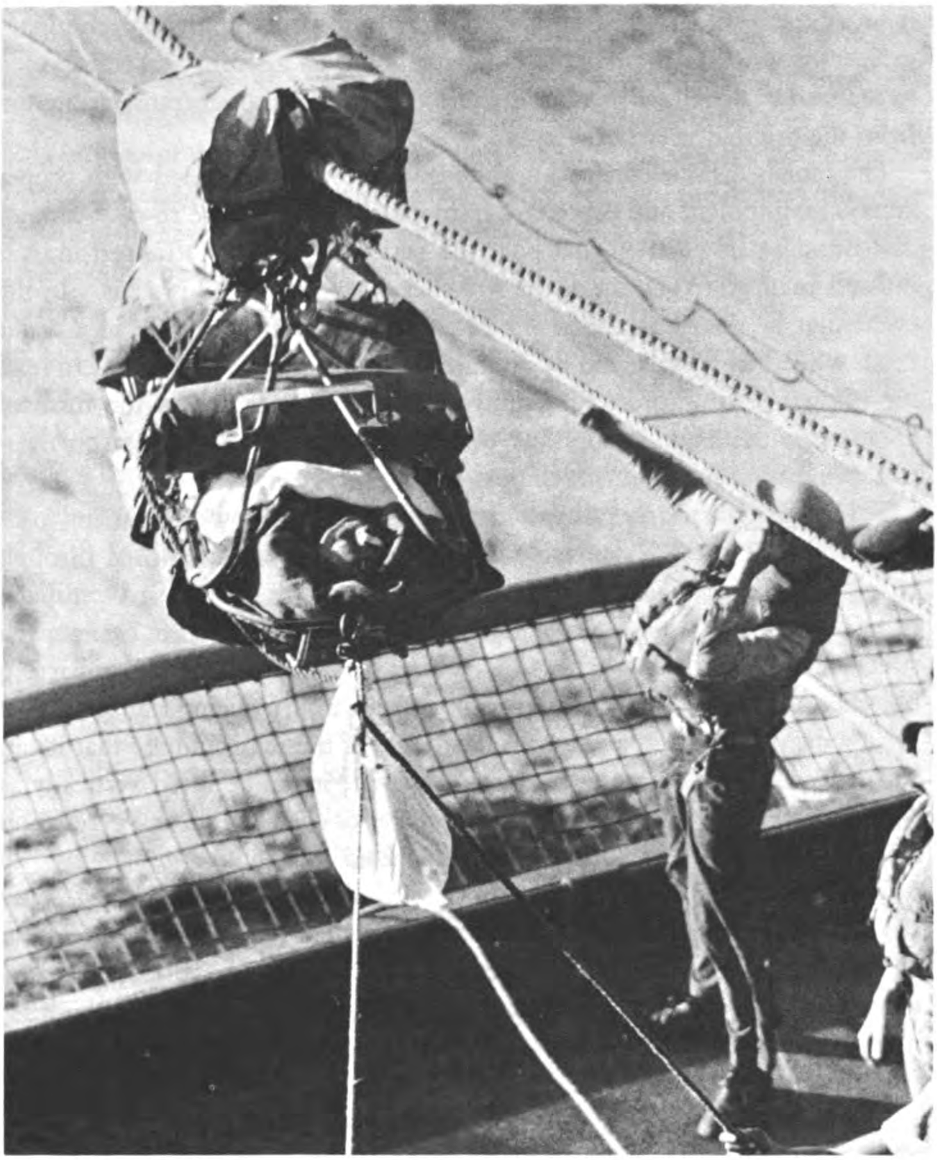

FIGURE 19–6. *Transfer of stretcher patient to carrier by highline. Note flotation equipment under top half of the Stokes stretcher—and protective frame* (USN PHOTO).

been widely used throughout the Navy for years. The prime advantage is that once inside it, the frame of the stretcher itself tends both to prevent further injury, and to prevent aggravation of existing injury. It is light and strong and almost always readily available. In most cases, once the patient is in a Stokes, he can be transported directly to sickbay, given preliminary care, transported to the flight deck, loaded into an aircraft, and transported to the facility which will render definitive care—all without transferring him from the original stretcher.

The Stokes is excellent for highline transfer when equipped with proper flotation equipment and protective frame as shown in figure 17-6. A major disadvantage, however, is that once the patient is properly strapped in, it is difficult for him to get out of the stretcher or even to free his arms. In the event that he goes into the water either on highline transfer or in a water crash while being evacuated by aircraft, he is thus unable to swim or to fend for himself. Some protection must therefore be provided in the form of flotation equipment (in highline transfer) and/or an attendant (on over-water flights) who can assist the patient in emergency escape.

The semirigid stretcher is specifically designed to allow the patient to be packaged in the smallest possible volume so that he can be moved through restricted openings with speed and facility. Greater care must be utilized in transporting a patient aboard ship in a semirigid stretcher, because the stretcher itself provides little, if any, protection from possible impacting objects, and does not prevent aggravation of the injury during transport to as great a degree as the Stokes. The advantages of the semirigid are that it can be used in spaces where the Stokes will not fit; it can be lifted vertically as through an escape trunk, and it is the stretcher of choice in patient evacuation from or through confined spaces and restricted passages.

The field stretcher or pole litter is carried aboard ship primarily for use by the Marines and/or landing party. It occupies less floor space than a Stokes and gives greater external protection than a semirigid. However, it is inadequate for patient transportation from confined spaces. It is *the* stretcher that must be used for MAC flights, and when a minimum number of transfers from one stretcher to another is desired during aeromedical evacuation, it should be utilized originally in evacuating the patient from the ship. If, however, the patient is to be flown from the carrier, the Stokes stretcher is preferred. With the field stretcher, an air mattress must be used to give comfort comparable to that of the Stokes.

Location of stretchers aboard ship is clearly delineated in the Battle Bill, which must be kept up to date by The Medical Officer. Periodic inspections under the supervision of The Medical Officer will insure that aged or missing handling lines and straps are replaced, that stretchers are in their designated locations, and are in usable condition.

The evacuation route selected for the patient often determines the stretcher type to be used. In cases of disasters or battle damage, routing for a patient in a particular area must be obtained from Damage Control Central (DCC). This unit is informed of the location and extent of all damage and is therefore able to select an evacuation route designed to avoid the hazard of injury to patient or crew and to retain maximum watertight integrity of the ship.

Generally speaking, one of the aircraft elevators should be used for transporting a litter patient to sickbay following an accident on or near the flight deck. Torpedo and weapons elevators may be used in emergencies.

In the acquisition phase, the key activities are training and inspection: (1) First aid training for the entire crew, especially medical officers and hospital corpsmen; (2) disaster training for the medical department, including the teaching of evacuation routes and discipline at the scene of disasters; and (3) inspections to insure the reliability and availability of emergency equipment. The Flight Surgeon, The Medical Officer and the medical department must be thoroughly prepared *prior to* the battle or disaster.

Other Vessel

Casualties occurring on small vessels usually receive first aid from other crewmembers, particularly if more than one casualty is present. The role of training is, therefore, fully as important as aboard a carrier.

Once first aid has been given and the patient evacuated to a suitable space where a medical department representative can examine and care for him adequately, his gross status must be determined—bones broken, pale or clammy skin, open wounds, etc.

The medical department representative then arranges a radio consultation with a medical officer on the nearest naval vessel with a medical officer aboard. (For this example, it is assumed that the casualty is aboard a destroyer which is in proximity to an aircraft carrier.) The medical officer's primary concerns are establishment of a working diagnosis; determination of the patient's condition as a result of the diagnosis; determination of the patient's treatment over the near term; and a decision as to whether the patient requires evacuation to the carrier.

If the medical officer recommends transfer of the patient to the carrier, he should at that time specify the recommended mode of transport so that the commanding officer may consider the risks and the alternatives. Any such transfer at sea entails certain risks to one or both vessels, to their crews, and to the casualty. The commanding officer has been carefully selected for his ability to make a thorough evaluation of all facts and to select the wisest course of action. The medical officer can assist him by (1) obtaining as many facts about the patient as possible during the radio consultation; (2) making as careful a differential diagnosis as possible with an estimate of the probability of each and a probable prognosis for each; and (3) presenting the case to the commanding officer as logically and succinctly as possible.

The casualty may be transferred from the destroyer to the carrier in a number of ways: by highline (stretcher or bosun's chair), by helicopter, or by small boat.

The patient will be considered either a stretcher or nonstretcher case. The stretcher case cannot ride in the bosun's chair but must be transported in the special Stokes stretcher equipped with flotation gear maintained aboard every carrier (see figure 19-6). Although stretcher cases can be transported by helicopter, such is not recommended except in extreme emergencies unless the helicopter can actually land on the smaller vessel.

Best surgical practice would probably have every case of suspected appendicitis travel via stretcher on the highline. In actual practice, however, many of them arrive without ceremony or previous consultation in the bosun's chair and seem to endure the trip just as well as in a stretcher—and they are safer en route.

With the patient's arrival aboard the carrier and in sickbay, the acquisition phase is completed. Thereafter, all casualties from all sources follow the same protocol, which is outlined below.

TRIAGE PHASE

When the patient, still on the stretcher, has reached the Emergency Room, the medical officer should quickly make a working diagnosis and preliminary measures of therapy should be started. If blood replacement appears indicated, a sample should be taken from the patient immediately so that members of the "walking blood bank" may be identified from the records and notified to report. It takes at least 45 minutes for them to make their way to sickbay, have their blood drawn, typed, and cross-matched, and only then can whole blood be administered to the casualty.

When several casualties with severe injuries arrive together or in rapid succession, the medical officer must make his working diagnoses rapidly in order to establish priorities for care, blood typing, surgery, and other services, in view of the limited resources available. X-rays and clinical laboratory examinations must be ordered at this time according to priority. Obviously, final decisions must be made by The Medical Officer in the case of multiple casualties with several medical officers attending them, since he must integrate the separate lists of priorities and establish final ones.

EMERGENCY TREATMENT PHASE

Air Transportability

After triage is completed and The Medical Officer knows the number of casualties and the nature and extent of injuries and illness, he must decide which patients can receive definitive treatment aboard and which must be evacuated to hospitals ashore. Of course, all patients continue to receive emergency care.

Transfer of patients from an aircraft carrier to shore facilities will in all likelihood be made using the C–2A COD (Carrier Onboard Delivery) aircraft shown in figure 19–7. This aircraft is pressurized, has a cruise speed close to 300 mph, and a normal range of 1,435 nautical miles. A litter kit is available to convert the C–2A rapidly for transfer of patients via Stokes litter. Figure 19–8 shows this conversion.

For emergency aeromedical evacuations, helicopters may be used. Figures 19–9 and 19–10 show the SH–3A *Sea King* and UH–2A *Seasprite* helicopters, respectively, either of which might be used for this purpose.

If The Medical Officer feels that some patients may be evacuated by air, their emergency treatment must reflect that possibility—that is, they must remain air-transportable, and any therapeutic measures undertaken must not render their travel by air impossible. While there are no absolute contraindications to AME, patients with any of the following conditions must be carefully evaluated for AME, particularly in unpressurized aircraft:

1. Respiratory embarrassment
2. Cardiac failure
3. Severe anemia, i.e., less than 2.5 million RBC per cu. mm or less than seven grams hemoglobin per 100 ml, estimated as nearly as possible to the time of flight.
4. Trapped gas within any of the body cavities, e.g., pneumothorax.

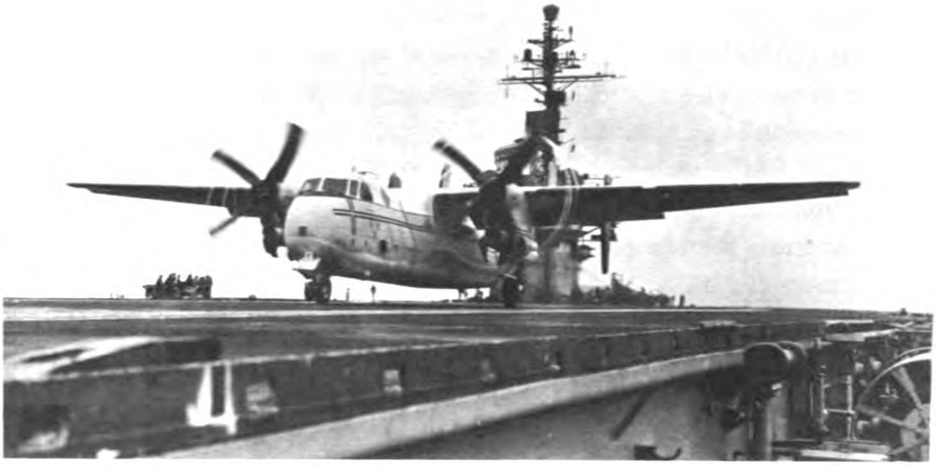

FIGURE 19–7. *C–2A COD (Carrier Onboard Delivery) aircraft used in transfer of patients from carrier to shore facilities* (GRUMMAN PHOTO).

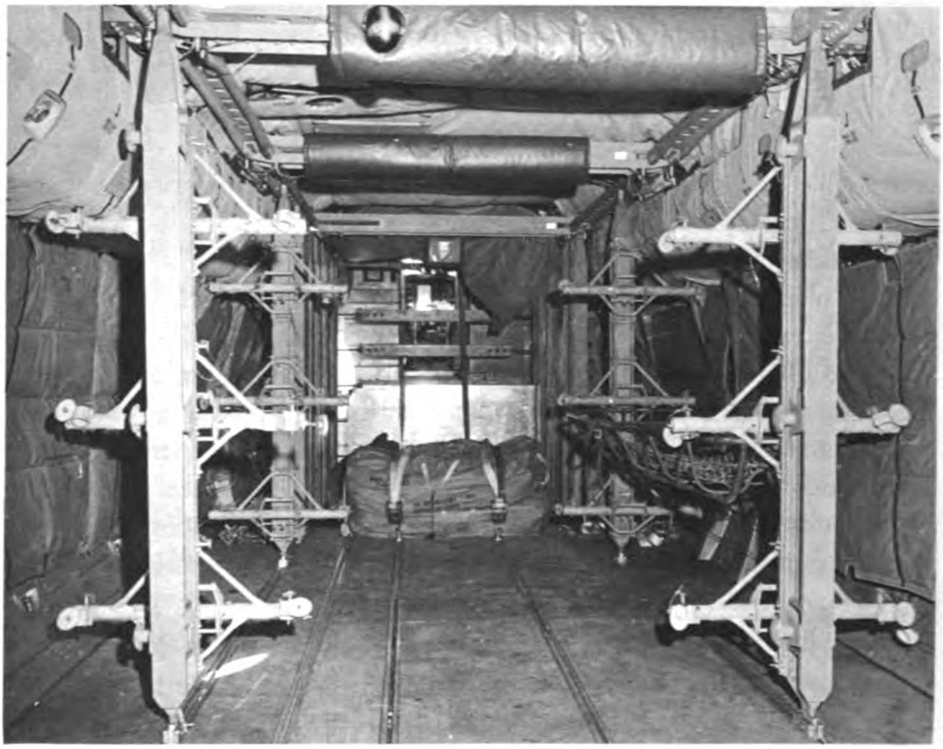

FIGURE 19–8. *Litter kit for rapid conversion of C–2A for patient transport via Stokes Litter.*

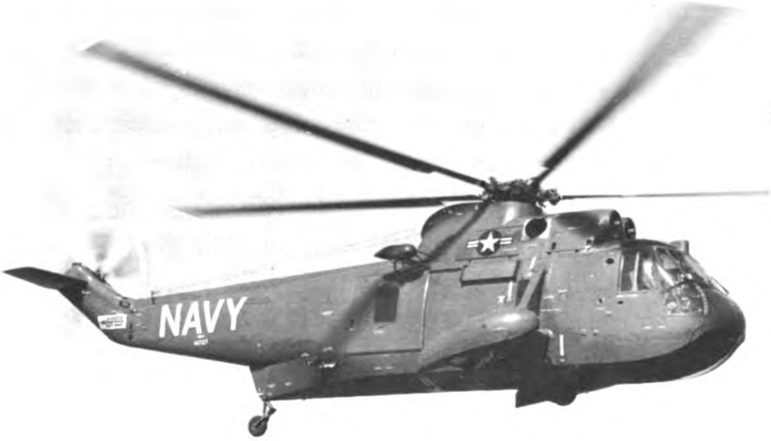

FIGURE 19–9. *SH–3A* Sea King *helicopter used for emergency aeromedical evacuation, although its primary mission is antisubmarine warfare* (SIKORSKY AIRCRAFT PHOTO).

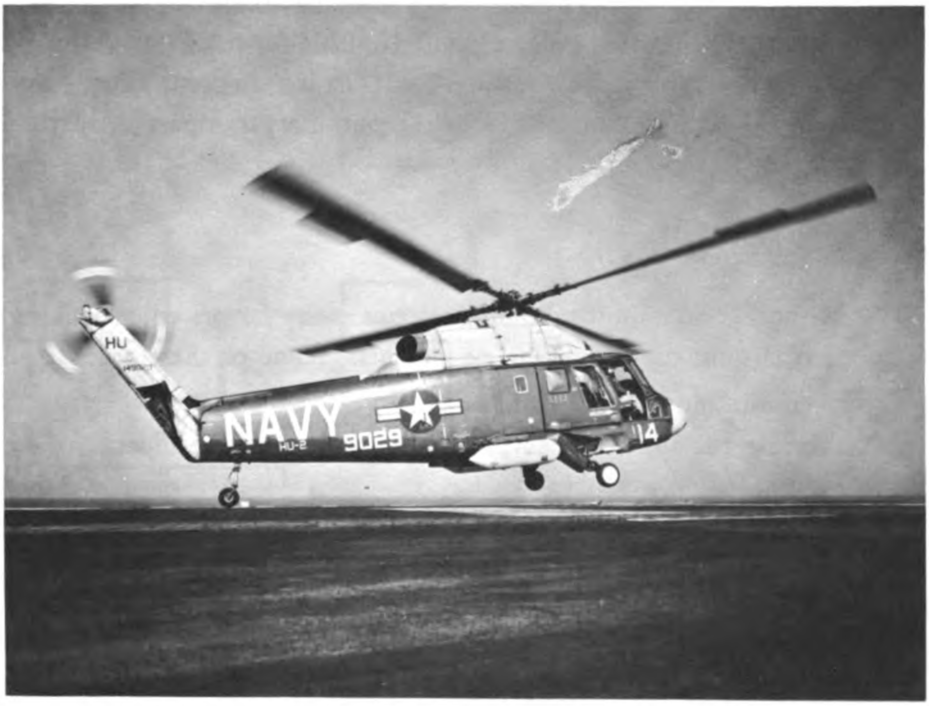

FIGURE 19–10. *UH–2A* Seasprite *helicopter used for emergency aeromedical evacuation* (USN PHOTO).

Such cases can all be moved by air with appropriate precautions. These might include inflight transfusion, maintenance of a low maximum inflight altitude by route selection, and furnishing of a well-trained attendant such as a Flight Surgeon, who can treat complications as they arise in flight.

Certain types of patients should be air evacuated only if there is no possible alternative:

1. Patients in the infectious stage of a serious communicable disease.
2. Moribund patients who are unlikely to survive the flight.
3. Patients whose upper and lower jaws are wired together, inasmuch as they need constant trained supervision, for the tie-wires must be cut immediately in the event of airsickness.

Other factors which affect air transportability of the patient include institution of: (1) therapy of any sort requiring maintenance of the patient in a bed, stretcher, or other conveyance which cannot be loaded through the hatch of the aircraft available, (2) orthopedic therapy which would preclude stretcher transport without further trauma to the injury site, and (3) therapeutic measures which, once applied, must be continuously used but cannot fit into or be utilized in the aircraft available for AME.

These problems rarely arise with MAC aircraft but do occur when Navy helicopters or COD aircraft are used for the preliminary transport phase from aircraft carriers.

Evacuation Decision

The Medical Officer must take into account many factors when making decisions regarding the evacuation of patients. Some of these are:

(1) Diagnosis and prognosis of patient.
(2) Treatment facilities available ashore vs. those available afloat.
(3) Transportation available ashore.
(4) Holding facilities available ashore.
(5) Diplomatic and legal aspects.
(6) Patient and crew safety in AME.

Diagnosis and Prognosis of Patient.—A patient who is going to die or lose a limb unless evacuated is one extreme. The other is the patient with an undiagnosed illness which might reflect a variation of normal or might be fatal if not treated early.

Facilities Available Ashore.—In order to determine whether there are adequate local hospitals or whether transfer by air to a distant hospital is required, the Flight Surgeon should have a comprehensive list of area hospitals, with the facilities and specialties available in each. It is helpful to have a list of nearby airfields and a description of airfield-hospital transportation arrangements for each, but these are seldom available. The Air Operations Officer can supply the names of nearby airfields and a description of airfield facilities. The Port Directory usually has a short description of local hospitals, their facilities, and specialities available at each. A list of U.S. military hospitals and medical facilities is generally available in foreign areas. Consular and embassy staffs can be of great assstance in detailing local medical facilities available and the diplomatic and administrative procedures required for admission of patients.

Possibilities for transfer are shown in figure 19–11. The shore facility may be a hospital or it may be an airfield from which further transportation can be arranged to a hospital.

When information about shore facilities has been received and evaluated, The Medical Officer must decide whether those facilities are sufficiently better than those on the carrier to justify evacuation. The carrier has a medium-

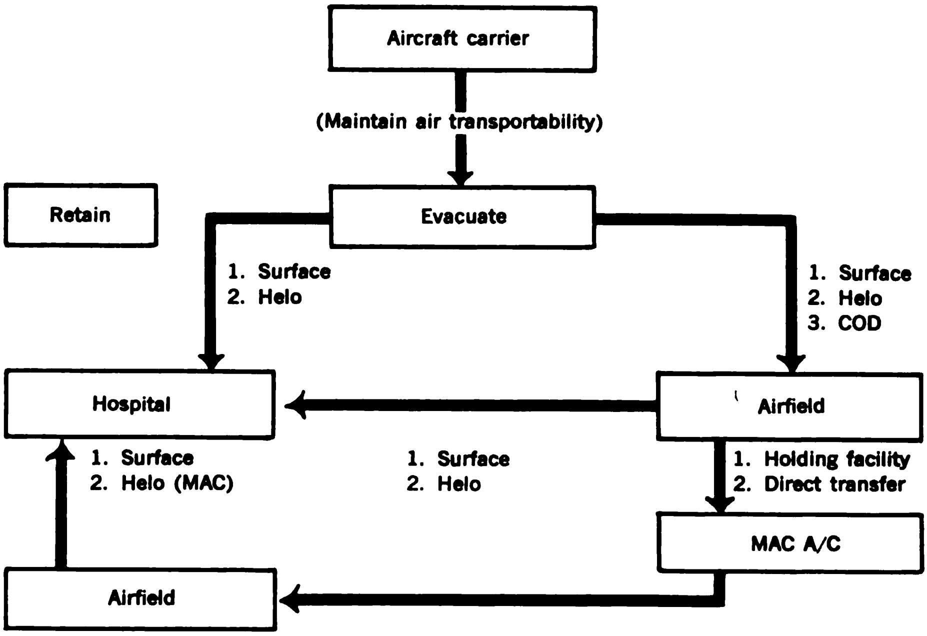

FIGURE 19–11. *Evacuation possibilities and modalities.*

sized hospital which is clean, dry, and air conditioned. Endemic disease is not a problem. There is unlimited potable water and sanitation is excellent. Facilities include a modern operating room, laboratory, X-ray with dark room, and physiotherapy equipment. There are large quantities of sterile supplies and an autoclave. At least four physicians (including a trained surgeon), at least three dental officers (who give invaluable assistance in emergencies), and a trained technical staff are available. Patients are secure from attack, and there is an almost unlimited supply of whole blood. In many areas of the world, such facilities are far better than anything ashore within thousands of miles.

If The Medical Officer decides that the patient must be hospitalized ashore, a decision must be made regarding the means of transportation.

Transportation Modalities.—If the hospital selected is within helicopter range of the ship, the patient may be transferred directly to it. Otherwise, suitable surface transportation may possibly be arranged by transferring the patient to a smaller vessel which takes him to the nearest port, with an ambulance completing the trip. A third possibility is that of having the patient transferred via COD aircraft to an airfield from which surface transportation to the hospital can be arranged.

Should the area be so remote that the patient must be transferred to an airfield by helicopter or COD and further transferred at the airfield to a MAC AME flight, there are several additional problems. If there is an armed forces holding facility at the airfield, the patient can be transferred ashore to that facility and maintained until the MAC flight arrives.

Holding Facilities.—Holding facilities are those (such as a small dispensary) which can provide nursing care, intravenous therapy and other emergency treatment, meals, shelter, hospital beds, etc., for a period of one or two days for patients en route to a hospital for definitive care but awaiting final airlift. If a facility adequate to handle the patient's problems is unavailable, arrangements must be made to have the evacuation flight from the ship meet the MAC AME flight and transfer the patient directly to the MAC aircraft. This type of transfer is tricky because it requires excellent communications, good weather, and good timing. This combination is not always available. Weather will not hurt the patient but it may prevent the MAC aircraft or the COD aircraft from landing. Communications in remote areas of the world differ greatly from those in Europe or the United States and are less reliable. Any delay in the rendezvous may cause a wait of hours in an aircraft, which is quite comfortable in the air but which becomes an oven or a freezer on the ground.

Diplomatic and Legal Factors.—Another difficulty which must be considered is national policy of the nation in which the hospital, airfield, or any en route airfield is located. Such policy may require diplomatic clearance and other administrative procedures which cannot be accomplished within the timeframe of the projected transfer. Again, United States Embassy and consular staffs can provide invaluable assistance.

Patient and Crew Safety in AME.—Safety of the crew of the helicopter or COD aircraft is a further consideration. It is far better to keep a questionable case on board than to subject the patient plus the aircraft crew to a flight made unsafe by inclement weather, enemy action, or mechanical unreliability of the aircraft. Aircrewmembers will volunteer for hazardous flights to attempt a lifesaving mission. The Flight Surgeon must realize this when deciding whether or not a case should be evacuated to preserve life or limb. He must consider the many facets of the problem mentioned here plus many others which will become apparent to him only on the scene and at the time. Each facet has value as a determinant.

Communications with MAC

When the decision to evacuate has been made and the commanding officer's approval for it has been granted, immediate attempts must be made to communicate with MAC, if a MAC airlift is required. Communications procedures are detailed in directives. General policy and information are contained in OPNAVINST 4630.9 series (Air Force Regulations 164-1 series). Specific information for the area of concern is contained in directives of the local area command. For example, in Northern Europe the Commander in Chief, U.S. Naval Forces Europe (CINCUSNAVEUR), has issued an instruction in the 6320.1 series, which sets forth procedures for aeromedical evacuation in that area. Such instructions normally include the name of the unit which will perform the evacuation, the procedure for establishing communication with the local Aeromedical Evacuation Control Officer, and other pertinent information.

Generally speaking, U.S. military shore stations in most areas overseas have a scheduled Aeromedical Evacuation flight which picks up patients routinely one or more times per week. Such information, including the schedule of the entire system is usually available thru the Aeromedical

Evacuation Control Officer of the local area, along with area supplements to Air Force Regulation 164-1 series (OPNAVINST 4630.9 series), which go into even greater detail on the AME procedures.

When a reply is received from MAC, the decision to evacuate may have to be modified in the light of facts contained in the reply. For example, if no aircraft is available or the weather will not permit sending one, or if the MAC AME flight cannot make satisfactory connections, The Medical Officer may decide to retain the patient and treat him aboard. MAC must, of course, be notified of such decisions.

Emergency Treatment and the Walking Blood Bank

While The Medical Officer is considering the question of aeromedical evacuation, the patient is given all necessary emergency treatment, subject to the limitations mentioned under air transportability.

One of the most important lifesaving measures is, of course, whole blood transfusion to replace blood loss. Since needs for whole blood are infrequent, it would be uneconomical to maintain a blood bank of the ordinary type. Instead, most carriers have a "walking" blood bank which is more suited to operational requirements.

The method of operation of the walking blood bank is simple. A log is kept by The Medical Officer listing a large number of personnel currently on board, by name, date, division, and phone number for each blood type. When blood is required, the laboratory technician hands a list of names to the Master-at-Arms, who is responsible for getting those personnel to sickbay as soon as possible. Blood is donated by these individuals and collected in blood transfusion bottles or bags while typing and cross-matching are performed. Soon after the blood donation is completed, the unit of blood, typed and cross-matched, is available for transfusion.

The problems associated with operation of the walking blood bank are maintaining currency of the log, keeping an adequate supply of blood collection sets and bottles on board, maintaining a supply of typing sera adequate for the number of blood bottles aboard, and finding a sufficient supply of donors for rare blood types. For example, there may be only three persons aboard with type AB Rh negative blood. If one such person requires a transfusion, only two units of blood are potentially available.

When a decision is made to evacuate a patient, blood is sent with him, as will be noted later.

INITIAL TRANSPORT PHASE

Patient Preparation

When communications with MAC or with a hospital have been established and definite plans for AME can be made, preparations for the MAC flight must be completed prior to departure from the ship, regardless of the transport modality to be used for the first few legs of the trip. Required administrative procedures, including patient classification, are defined in OPNAVINST 4630.9 series, BUMEDINST 6320.1 series, BUMEDINST 4650.2 series, and BUMEDINST 6700.2 series.

Required patient preparations include the following:

1. Prepare patient's identity card to include information as to special treatment required en route, restrictions as to altitude, baggage check, and any other special information, and attach to the patient, as noted in BUMEDINST 4650.2 series.
2. Provide special medications and materials required by the patient en route, including drugs not normally carried by MAC medical attendants. All drugs should be labeled with name and strength of drug, dosage, and directions for its administration.
3. Give preflight medications according to current directives or at the discretion of The Medical Officer.
4. Apply clean dressings as near the time of departure as practicable, particularly on colostomies, draining wounds, burns, and pressure ulcers. Where frequent dressings are required, provide the patient with a 24-hour supply. Colostomies require more frequent dressings en route because of increased intestinal gas pressure due to altitude.
5. Give intravenous fluids, when required, as close to the time of departure from the carrier as possible. Keep cutdowns open with slow drip of five percent dextrose in water.
6. Give enema, when required, as close as possible to time of departure from the carrier. There are no facilities for giving enemas in flight.
7. Apply indwelling catheter in cases requiring frequent catheterizations. In cases requiring irrigation, provide a supply of the solution to accompany the patient.
8. Transfuse patients with low hemoglobin or an RBC count of less than 3,500,000. These patients are not acceptable on a MAC flight until danger of reaction has passed.

9. Replace maxillary fixation with rubber bands. This is to avoid pulmonary aspiration in case the patient becomes airsick.

10. Bivalve casts which have been applied within 24 hours of departure from the carrier.

11. Furnish masks to pulmonary tuberculosis cases unless the record definitely states that the infection is arrested or nonactive.

12. Apply supports for extremity paralysis cases to prevent drop.

13. Sedate neuropsychiatric cases Class 1A and 1B and deliver to aircraft on a litter in pajamas. The sedation of choice is oral or intramuscular sodium amytal, which is the sedative used en route. MAC medical attendants are better able to judge the depth of sedation if the preflight medication is the same drug.

14. Apply restraints to all Class 1A patients, and to any Class 1B patients who may be combative, suicidal, or violent, and to any doubtful case.

15. Brief the AME medical attendants with respect to preflight medications, characteristics of psychotic patients, particularly in regard to combative, assaultive, or suicidal trends, and in any special treatment or medication required by any of the patients.

16. Provide food (including special diets) that is not ordinarily carried on the type of AME flight contemplated.

In addition, litters must be prepared for expected weather conditions on the flight deck and at the first transfer point (holding facility, hospital, or airfield) as necessary.

The patient's personal effects must be collected and packed, his records must be brought up to date and prepared for transfer, and a summary of his present illness, at least, must accompany him in the Health Record.

The Flight Surgeon must gather information required for patient preparation and en route support. Some of the information required, and the reasons for it, follow:

1. Aircraft pressurization capability.

2. Flight altitudes en route. When these two facts are known, the Flight Surgeon can recommend the maximum cabin altitude to which the patient should be subjected.

3. Availability of emergency oxygen equipment. If oxygen might be required but is not available aboard the aircraft, it can be furnished from the ship's supplies.

4. Type of attendant required. Normally a trained and experienced hospital corpsman will accompany the patient, but if the patient's condition requires it, a Flight Surgeon will attend him.

5. Expected flight duration, with ETD and ETA. The quantities of required medical supplies; meals for inflight feeding of patient, attendant, and aircrew; consumables such as oxygen, coffee, and drinking water, and arrangements for cabin lighting all hinge on this information.

6. Expected destination weather at ETA. Clothing, blankets, and other arrangements for thermal comfort may be required en route and at destination.

7. Routes of emergency exit and emergency equipment on the aircraft. The attendant *and the patient* need to have this information in the event of a water crash. Arrangements must be made for emergency flotation either of the patient or of the entire man-stretcher package. Of course, all possibilities cannot be covered, and there is little chance for the bundled-up, unconscious patient strapped into a Stokes.

8. En route stops planned, holding facilities available, and diplomatic procedures required. Necessary visas, immunization certificate, and other necessary documents must be provided for patient, attendant, and aircrew.

9. Location of attachment points for IV fluid bottles, stretcher, and catheter drainage receptacles. Such points may not be available at the location selected for the stretcher; also, in the event of light failure, the attendant or Flight Surgeon must know where to look.

The Air Operations Officer can supply most of this information. The remainder can be obtained both by flying routinely in the aircraft to become familiar with it and by examining the aircraft interior prior to an AME flight.

Another necessary preparation is the collection and chilling of requisite amounts of whole blood. Emergency chilling can be accomplished by using ice cubes in plastic shirt bags obtainable from the laundry. This blood may save the patient's life if he starts hemorrhaging at any point in the trip, even after landing at the destination, since typing and cross-matching have already been performed, and it is available for instant use.

Transport from Sickbay and Aircraft Loading

Careless stretcher bearers can do considerable damage to carefully prepared patients on the short trip up a ladder to the deck-edge elevator. The Flight Surgeon should supervise the whole move, since his presence calms both the patient *and* the stretcher bearers. Tight strapping of the patient to the litter will assist in preventing trauma while loading through a narrow hatch or with the litter in an unusual attitude. After loading, the Flight Surgeon should check the provisions for emergency escape for both attendant and patient, and assure himself that neither will be injured in a normal takeoff and landing.

Emergency Patient Care En Route

In selected cases, the Flight Surgeon will accompany the patient at least to the first transfer point, in order to be able to detect and treat medical emergencies en route. When the Flight Surgeon deems it necessary, he should remain with the patient until he is out of danger or has been transferred to competent medical personnel.

A patient who might go into shock en route, but who does not at present require transfusion, should have an indwelling venous catheter inserted prior to transfer. It should be kept open with IV dextrose in water administered through a blood transfusion IV set which has been "pressurized" by taping all the points which might separate should blood have to be administered under pressure.

Neither IV fluids nor blood run as freely at altitude as at sea level, since the IV motive force of atmospheric pressure decreases with ascent. Therefore, if blood must be given rapidly, additional pressure must be supplied, usually by using a rubber bulb. If the flight is in an operational aircraft (as is usually the case), the rear compartment lighting is usually poor, there is insufficient space, and most normal medical arrangements are improvised. The wise Flight Surgeon will prepare for the worst prior to leaving the ship, because there is no place to turn at 10,000 feet at night, two hours from destination, with a patient who suddenly goes into shock or starts massive hemorrhage.

As noted previously, typed and cross-matched blood should be carried with the patient. If not required in flight, it may be useful later at the hospital or on the MAC flight.

The pilot should be kept informed of the patient's condition so that he can adjust the flight plan to assist the Flight Surgeon. In some cases, he can find a less turbulent altitude. In others, he can decrease altitude or increase airspeed. He can radio ahead for ambulances, surgical teams, unusual supplies, or other ground assistance so that it will be waiting when the aircraft lands. He may be able to land at a nearby airfield with faster access to a hospital than available at the original destination.

Holding Facilities at Transfer Point

Holding facilities, as the term is used here, do not include casualty staging activities established by MAC in the United States and elsewhere. Such MAC activities are generally well run and more than adequate. The Navy problem is in obtaining similar services from much more modest installations wherever in the world the casualty occurs. The Flight Surgeon must improvise to obtain adequate care for his patient if his examination of the available facilities shows them to be inadequate.

The adequacy of the holding facilities has previously been mentioned as an important factor in deciding whether to attempt a direct transfer from the ship's COD or helicopter to the MAC AME flight at the airfield or to transfer the patient to the holding facility. Information available to the Flight Surgeon aboard ship about the holding facility is frequently inadequate. In such case, it is far wiser to plan a direct transfer.

Patient, Records, and Property Transfer

The initial transport phase ends when the patient has been delivered to the hosiptal or to the MAC AME flight, the necessary records have been transferred, and the ship's property signed for. BUMEDINST 6700.2 series covers the property exchange and accounting, and the Flight Surgeon and/or medical attendant should familiarize himself with these procedures prior to leaving the carrier.

FINAL TRANSPORT PHASE

The final transport phase is entirely under the control of MAC, but proper advance planning should hold problems during this phase to a minimum.

REFERENCES

Department of the Navy, Bureau of Medicine and Surgery. Medical regulating to and within the continental United States. BUMEDINST 6320.1 series.

Department of the Navy, Bureau of Medicine and Surgery. Patient evacuation manifest, DD Form 601, modification in the use of for aeromedical evacuation of patients by MATS. BUMEDINST 4650.2, Suppl. 1.

Department of the Navy, Bureau of Medicine and Surgery. Property exchange accounting in evacuation of patients. BUMEDINST 6700.2 series.

Department of the Navy, Office of the Chief of Naval Operations. Worldwide aeromedical evacuation. OPNAVINST 4630.9 series.

CHAPTER 20

Preventive Medicine

RESPONSIBILITIES OF THE FLIGHT SURGEON
PUBLIC HEALTH PROBLEMS IN THE NAVAL SERVICE
    Communicable Disease Control
    Quarantine
    Immunization Procedures
SUPPORT SERVICES
    Preventive Medicine Units
    Other Armed Forces Medical Facilities
REFERENCES

RESPONSIBILITIES OF THE FLIGHT SURGEON

ONE OF THE primary missions of the Medical Department is that of safeguarding the health of Navy and Marine Corps personnel. This mission is carried out largely through a program of preventive medicine which focuses upon the preservation of health and maximum effectiveness of the individual. In the area of preventive medicine, the medical officer is responsible for maintaining surveillance in all matters pertaining to health, for advising the commanding officer of the existence of conditions that may have a deleterious effect on the health of personnel, and for recommending to the commanding officer the implementation of such measures as may be necessary to maintain the health of the command.

Information concerning the general and specific preventive medicine authority and responsibilities of medical officers in the U.S. Navy is available in a number of sources which are readily available at each command. The following references are of particular value and the Flight Surgeon should familiarize himself with their contents.

1. *Manual of Naval Preventive Medicine.* This manual points out in detail the official methods of dealing with public health problems in the naval

service. It should be the first reference consulted when a question arises concerning such problems as environmental sanitation, food and water sanitation, garbage and refuse disposal, insect and rodent control, and ventilation.

2. *Manual of the Medical Department* (*MMD*). In addition to Chapter 22, which contains the General Provisions Concerning Preventive Medicine, Articles 3–2, 3–4, 3–12, and 23–124 are pertinent.

3. *Bureau of Supplies and Accounts Manual,* Vol. IV. Sections of this publication deal with inspection of subsistence items by Medical Department representatives (41297), and testing facilities for food products (41298).

4. *General Orders.* No. 18 concerns the repression of prostitution and the control of venereal diseases. No. 20 concerns quarantine regulations for vessels and aircraft of the armed forces.

5. *Navy Directives System.* Many specific subjects are covered by Instructions or Notices promulgated by the various bureaus of the Navy Department. Those in the 6000 series are issued by the Bureau of Medicine and Surgery. Series 6200 usually concerns preventive medicine. The Flight Surgeon should review these directives to learn what the current policy is regarding various medical matters.

6. *Navy Regulations* (*NR*). Articles 0969, 0970, and 1981 define certain of the duties of Medical Department personnel relating to preventive medicine.

In addition to these directives, a great many other publications are available concerning every possible aspect of preventive medicine. As the need becomes apparent, the Flight Surgeon should consider procuring supporting publications for reference and guidance. If revisions and supplements are published, he should insure that his department is on the distribution list to receive current addenda. Also, the Bureau of Medicine and Surgery may be contacted for assistance in procuring publications.

The Flight Surgeon should also be familiar with public health regulations and practices in communities adjacent to naval installations. He should maintain liaison with local public health authorities such that he can apprise his commanding officer of conditions that may affect the health of personnel residing in or frequenting these areas.

PUBLIC HEALTH PROBLEMS IN THE NAVAL SERVICE

This section covers those areas not considered in the *Manual of Naval Preventive Medicine.*

Communicable Disease Control

The Medical Department must constantly be alert for the early detection of infectious diseases. The department is responsible for recommending to the commanding officer those measures necessary to prevent the spread of communicable disease, including the institution of quarantine of personnel.

Venereal disease is one particular area of communicable disease that, like the others, is better handled by prevention. It is the duty of the Flight Surgeon to present factual information to those in his care in lectures and by any other media available.

MMD states that the current edition of the following publication may be used by medical officers as a guide for communicable disease control: *Control of Communicable Diseases in Man,* published by The American Public Health Association, 1790 Broadway, New York, N.Y. 10019. This authoritative publication is updated annually and identifies each disease as to its clinical nature, laboratory diagnosis, and differential diagnosis, as well as the infectious agent, reservoir, mode of transmission, incubation period, and period of communicability. Methods of control are described.

It is important that medical officers cooperate with Federal, State, and local health agencies regarding the prevention and reporting of communicable diseases. Chapter 23, *MMD*, presents instructions for the preparation and submission of routine reports and notices concerning the presence of communicable disease and other matters in the field of preventive medicine.

Quarantine

Quarantine procedures prevent the introduction and dissemination domestically or elsewhere of human diseases, animal diseases, or plant diseases. General Order No. 20, "Medical and Agricultural Foreign and Domestic Quarantine Regulations for Vessels, Aircraft, and Other Transport of the Armed Forces," contains the basic regulations and detailed instructions concerning such procedures.

Flight Surgeons should be well informed concerning current quarantine regulations and instructions. It is the responsibility of the Medical Department to insure compliance with these regulations by advising and making recommendations to the commanding officer. Since quarantine regulations may differ from port to port, the Medical Department must determine in advance the particular requirements for entry and assure that necessary quarantine declarations are delivered to Port Authorities. Information concerning requirements for entry into foreign ports is available in the Port Directory which is maintained in the administrative office of the Senior Officer Present Afloat (SOPA).

There are four classes of quarantine authority with which the Navy must comply:

1. *United States, Its Territories and Possessions.* Federal Statute assigns quarantine authority and responsibility to the U.S. Public Health Service (human diseases), the Department of Agriculture (plant and animal diseases) and the Department of the Interior (wildlife).
2. *Cities, Counties, and States.* Some locations have promulgated quarantine regulations in addition to those of the Federal agencies.
3. *Foreign Countries.* Compliance with the laws of foreign countries is mandatory.
4. *Outside the United States, Its Territories, and Possessions.* At naval establishments where there is neither Federal nor civil authority, quarantine responsibility devolves upon the naval district commandant or the area or base commander.

By keeping well informed concerning current quarantine regulations and assuring full compliance with all quarantine authority, the Medical Department can minimize delays associated with port arrivals and departures. Such delays could be most disruptive to operations and could cause needless inconvenience to a great many persons.

Quarantinable Human Diseases.—There are six diseases classified as quarantinable by international agreement. These are:

1. Cholera
2. Plague
3. Louse-borne typhus
4. Smallpox
5. Yellow fever
6. Louse-borne relapsing fever

Immunization Procedures

Chapter 22, Section VIII, *Manual of the Medical Department* and BUMED directives in the 6230.1 series provide guidelines for immunization of naval and Marine Corps personnel. The medical officer having custody of a health record is responsible for immunization of the person for whom the health record was issued. *MMD* requires routine immunizations at stated intervals and details the records which must be maintained.

As a rule, the recordkeeping associated with immunizations, especially when given en masse, is fully as important as the administration of the immunizing agent itself. As a result of clerical omissions, a certain percentage of the population may remain unimmunized and thus serve as a reservoir of disease. Just as importantly, failure to record immunizations promptly and accurately may arouse the most primitive emotions in those personnel required, because of clerical error, to undergo a second immunization. Rapport between a Flight Surgeon and his unit, which is essential to the practice of aerospace medicine, may be severely impaired by just such a small matter.

If, despite careful checking and cross-checking of immunizations and their recording, an occasional error occurs, there will be few objections. If, however, these procedures are handled casually, an unfortunate impression of the medical department will be created.

The *Manual of the Medical Department* states that "Naval and Marine Corps personnel shall not be transferred from a training station, barracks, receiving station, or other rendezvous except in emergency until required immunizations have been given *and are recorded* in their Health Records." If this directive is to be followed without loss of time from duty, medical staff members charged with responsibility for maintaining immunization records must be informed by the unit personnel officer as soon as *he* receives a copy of transfer orders for any individual in the unit. In this way, orderly scheduling of immunizations can be accomplished.

Requirements for specific immunizations are determined, in part, by the geographic area of concern. Scheduling of immunizations for entire units should be arranged so that a high incidence of immunization reaction will not paralyze the unit. Immunizations are frequently scheduled for and given on payday so as to insure maximum unit attendance. Determination of which individuals missed the unit schedule requires monotonous and time-consuming checking of records, and such needless effort should be kept to an irreducible minimum. Each person, however, *must* be immunized to guarantee unit effectiveness in the event of exposure to biological warfare agents or to contagious diseases peculiar to the area of operations.

Scheduling of aviators' immunizations must be coordinated with the unit Commanding Officer or Operations Officer. If possible, the entire group can be immunized prior to a layoff period so that no time will be lost from the flight schedule. BUMEDINST 6230.1 series directs that, "All aircrews will be grounded a minimum of 8 hours (24 hours if not detrimental to mission) after receiving any immunization except oral polio virus vaccine and smallpox vaccine."

If group immunization is not possible, aviators' immunizations must be scheduled on an individual basis to minimze lost time. Aviators demonstrating immunization reactions to any immunizing agent must be grounded until completely recovered. This is another reason for administering immunizations prior to a layoff period, since it encourages anyone who suffers a reaction to report it to the Flight Surgeon by (or before) the end of the layoff period, before the next flight schedule is drawn up.

Upon initially reporting to their units, aviators are usually required to receive a flight clearance notice from the Flight Surgeon (on NAVMED Form 1381). One reason for this requirement is that it allows the Flight Surgeon to examine the health record for (1) health conditions for which the aviator might require followup, (2) status of required aviators physiology training as reported on SF 1349, so that he can be scheduled for completion of requirements at the earliest possible date, and (3) immunization status, as carried in the Health Record on SF 601, so that he can receive, or be scheduled for, any required immunizations prior to commencing his flying duties.

Maintenance immunizations within a unit must be scheduled in advance. Requirements for maintenance immunizations are determined during the annual Health Record Review.

MMD 16–51 requires that naval and Marine Corps personnel traveling to foreign countries under the cognizance of the Navy Department shall be immunized as indicated in Chapter 22, *MMD,* and in current BUMED directives, *and* that DD Form 737, *Department of Defense Immunization Certificate,* properly executed and authenticated, shall be in their possession *prior to embarkation.*

The Flight Surgeon should advise all aviators to carry the DD 737 with them at all times when flying outside the United States. The reason is that some nations require presentation of this form or else immediate vaccination or other immunization, if the aviator enters the country under *any* circumstances. For example, if an aviator flying a training mission from a carrier must make an unexpected emergency landing ashore, he may well be greeted by a health officer as he deplanes. If he does not have the DD Form

737, he will receive the immunizations required by that nation and will thereby be grounded for eight hours, as noted in BUMEDINST 6230.1 series. The resulting delay in returning to his ship may cause scheduling problems, which are easily avoided simply by requiring each aviator to carry the form. It is true that many nations tacitly ignore the regulations, but they are not required to do so by any treaty provisions.

SUPPORT SERVICES

At times the Flight Surgeon may find that the problems of implementing and monitoring an effective preventive medicine program within his activity are extremely complex, demanding much in the way of highly specialized skills, knowledges, and procedures. There is usually an industrial hygienist available at each major air station and a Preventive Medicine Technician aboard most carriers to render assistance as needs arise. The Flight Surgeon should request the services of these personnel in carrying out his preventive medicine program. There may be times, however, when the problems at hand will require technical assistance beyond that available within the command.

Preventive Medicine Units

To provide specialized consultation, advice, and recommendations in matters of preventive medicine and environmental health, Navy Preventive Medicine Units have been established in support of naval shore activities and Fleet units in a number of assigned areas. In general, the PMUs conduct epidemiological investigations, special sanitary surveys, and performs supporting laboratory examinations. They also provide training in preventive medicine procedures and techniques, perform quarantine inspections, and visit ships and stations to provide guidance for the control of conditions affecting health. In summary, the PMU is available to provide preventive medicine services, when required, that are beyond the capabilities of activities requesting them. They do not, however, perform routine clinical laboratory tests. BUMED Instructions in the 5450 and 6200 series should be reviewed for additional information.

Also available for assistance in certain areas of preventive medicine are the Navy Disease Vector Control Centers. It is the mission of these units to provide technical and specialized services in the fields of vector prevention and control as may be authorized and directed.

It would be of great benefit for the Flight Surgeon to familiarize himself, either by personal visit or correspondence, with the facilities and services offered by these units. The following is a list of the units and their locations:

Navy Preventive Medicine Unit No. 2
Norfolk, Va. 23511
(This unit provides services for the
entire east coast of the United States.)

Navy Preventive Medicine Unit No. 5
Naval Hospital
San Diego, Calif. 92134

U.S. Navy Preventive Medicine Unit No. 6
Pearl Harbor Naval Shipyard
Pearl Harbor, Hawaii
(Mail Address)
U.S. Navy Preventive Medicine Unit No. 6
Box 110
FPO San Francisco 96610
U.S. Navy Preventive Medicine Unit No. 7
Naples, Italy
(Mail Address)
U.S. Navy Preventive Medicine Unit No. 7
FPO New York 09521

Navy Disease Vector Control Center
Naval Air Station
Alameda, Calif. 94501

Navy Disease Vector Control Center
Naval Air Station, Box 43
Jacksonville, Fla. 32212

Requests for the services of these units will usually be governed by geographic location and by facilities and services available. In an emergency, a direct telephone or telegraphic request may be made, with later written confirmation submitted through channels. Current BUMED Instructions in the 6200 series should be consulted regarding the official channels for requesting services which are not of an emergency nature. It should be noted that even if a given unit cannot provide the requested service itself, it is well prepared to offer assistance and guidance concerning where and how to secure help.

For a better understanding of the operation of the PMU, the Flight Surgeon should view the recent film, "The Preventive Medicine Unit," MM-10045. In addition, it is advisable for the Flight Surgeon to assure that his medical department is on the mailing list for papers such as health notes or monthly newsletters published by several of the Preventive Medicine Units.

In addition to the PMU, the Flight Surgeon may get assistance in dealing with specialized problems of preventive medicine from:

Naval Medical School
National Naval Medical Center
Bethesda, Md. 20014

Other Armed Forces Medical Facilities

There are a number of armed forces laboratories and medical service facilities throughout the country. Should a Flight Surgeon wish to know what facilities are available in his area, and their specific capabilities, he can secure such information from the local Navy Medical Officer.

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Fleming, A. J., D'Alonzo, C. A. (Eds.), & Zapp, J. A. (Assoc. Ed.). *Modern occupational medicine.* Philadelphia: Lea & Febiger, 1960.

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CHAPTER 21

Space Flight

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- Medical Sciences in Space Operations

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INTRODUCTION

REFLECTING on man's role in space, the late Dr. Hugh Dryden[1] said, "None of us knows what the final destiny of man may be, or if there is any end to his capacity for growth and adaptation. Wherever this venture leads us, I am convinced that the power to leave the earth—to travel where we will in space and to return at will—marks the opening of a brilliant new stage in man's evolution." To date, our astronauts have demonstrated man's capacity to live and work for fourteen days in space. Before the next decade, they will demonstrate their ability to live on the moon. Beyond that, there are the planets and the stars. With the spacecraft, man has unlocked the door to the solar system. He now is presented with closeup views of the moon and planets and, as shown in figure 21–1, can even view the earth as it would be seen by the inhabitants of another celestial body.

From information obtained through space probes and from observations from earth, much now is known about the solar system and the planets within it. Table 21–1 shows the orbital characteristics of the planets and describes their physical properties. The full size of the solar system, which is almost infinitely small in relation to other space systems, is evidenced by the distance between Pluto, the farthest planet, and the sun, a matter of 4,680,000,000 miles when Pluto is at its greatest distance.

Celestial Mechanics

The principles underlying space travel are quite different from those of travel within the earth's atmosphere. A full appreciation of the human problems involved in interplanetary missions can be aided by a brief review of the guidance and navigational techniques to be used.

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[1] Dr. Hugh L. Dryden was Deputy Administrator of the National Aeronautics and Space Administration from its creation by Congress in October 1958 until his death on December 2, 1965. Dr. Dryden was internationally recognized for his scientific contributions to fluid mechanics and boundary layer phenomena, and was highly honored for his leadership of research and development associated with aeronautics and astronautics. He generally was regarded as the man who guided the United States into the Space Age. Dr. Dryden became Director of Research of the National Advisory Committee for Aeronautics (NACA) in September 1947, and was named Director of NACA in May 1949, serving until it was superseded by NASA in 1958.

FIGURE 21–1. *The earth as seen during lunar orbital flight* (NASA PHOTO).

The concept of "straightline distance" has virtually no meaning when applied to the problems of traveling from one celestial body to another. In many respects, the manned mission to the moon represents the most complex guidance problem ever undertaken by man, and it's solution draws heavily on the fundamental principles of the sciences of mechanics, physics, and mathematics (Funk, 1964). There are, in addition, many unique problems which have arisen and which require a special guidance technology.

Four primary problems have to be solved in order to guide a spacecraft to its destination:

1. Determine the present position and velocity (state vector) of the spacecraft.
2. Predict the future path of the spacecraft from the measured state vector and evaluate the flightpath error.
3. Calculate the correction to the present state vector required to correct the present flightpath to the desired flightpath.
4. Execute the required propulsion maneuver to change the flightpath.

TABLE 21-1.—*Solar and Planetary Orbital and Physical Data*

Body	Mean heliocentric distance A.V. (10^6 miles)	Sidereal period, days (years)	Rotation period	Closest approach to earth (10^6 miles)	Number of satellites	Mass earth = 1 (Kg)	Diam, earth = 1 (miles)	Density, earth = 1 (water = 1)	Gravity earth = 1 (cm/sec^2)	Surface temp. °C
Sun	0 (0)	....	27 days (av)	93 (av)	9	333,400 (1.989×10^{30})	109 (864,600)	0.25 (1.41)	28 2.74×10^4	5,727
Moon	1 (92.90)	29.53	27.32 days (about earth)	0.221	....	0.0123 (7.35×10^{22})	0.272 (2,160)	0.605 (3.34)	0.165 (162)	130 (max) <-168 (min)
Mercury	0.3871 (35.96)	87.97	88 days	50	0	0.055 (3.3×10^{23})	0.379 (3,008)	0.60 (5.46)	0.38 (372)	395 max
Venus	0.7233 (67.20)	225	240 days	26	0	0.813 (4.86×10^{24})	0.955 (7,567)	0.996 (5.5)	0.891 (874.3)	307 av
Earth	1.0000 (92.90)	365.256	23 hours 56 min	....	1	1 (5.966×10^{24})	1 (7,927)	1 (5.52)	1 (981.3)	15
Mars	1.5237 (141.6)	686.980	1.03 days	34.6	2	0.107 (6.4×10^{23})	0.523 (4,141)	0.80 (4.12)	0.39 (382.7)	25 max −13 min
Jupiter	5.203 (483.3)	(11.862)	.41 days	367	12	316.7 (1.89×10^{27})	11.2 (86,700)	0.24 (1.33)	2.65 (2,600)	−130
Saturn	9.54 (886.1)	(29.458)	.43 days	745	9	94.7 (5.6×10^{26})	9.47 (75,065)	0.13 (0.71)	1.17 (1,148)	−150
Uramus	19.19 (1,783)	(84.015)	.45 days	1,606	5	14.58 (8.7×10^{25})	3.9 (30,900)	0.28 (1.56)	1.05 10.30	−130 (max)
Neptune	30.071 (2,794)	(164.788)	.66 days	2,700	2	17.0 (1.02×10^{25})	4.16 (32,900)	0.45 (2.47)	1.23 (1,207)	−160 (max)
Pluto	39.5177 (3,670)	(247.697)	....	....	....	<0.1 (5.966×10^{23})	0.45 (3,600)	0.99 $<(5.5)$	<0.5 (490)	−190 (max)

(Data from "Space and Astronomy" by C. D. Graves, in *System Engineering Handbook*, R. E. Machol (Ed.), copyright 1965, McGraw-Hill; and from *Handbook of Geophysics and Space Environments*, S. L. Valley (Scientific Ed.), copyright 1965, McGraw-Hill. Used by permission of McGraw-Hill Book Company.)

Problem 1 is the navigation problem. Problems 2 and 3 are the guidance problem, and problem 4, the control problem.

There are a number of critical guidance problems in the Apollo Lunar mission, as shown in figure 21-2 (Chamberlin, 1964). After initial insertion into a parking orbit, the launch vehicle injects the spacecraft into a translunar orbit. The guidance system must provide midcourse corrections in order to shape the trajectory and to compensate for errors so as to reach the vicinity of the moon. It then must establish thrusting requirements for the lunar orbit. After establishing this orbit, the appropriate signals must be given to the Lunar Module to separate and be braked from orbit. Guidance must then be given for the final approach and landing. After a short stay on the moon's surface, the ascent stage of the Lunar Module must be injected into a lunar orbit such that it can rendezvous with the command module. After the transfer of astronauts is complete, transearth injection of the Apollo spacecraft is effected. Midcourse corrections must again be computed to reach the required earth reentry corridor. The onboard guidance then provides the information required to land at the selected landing area.

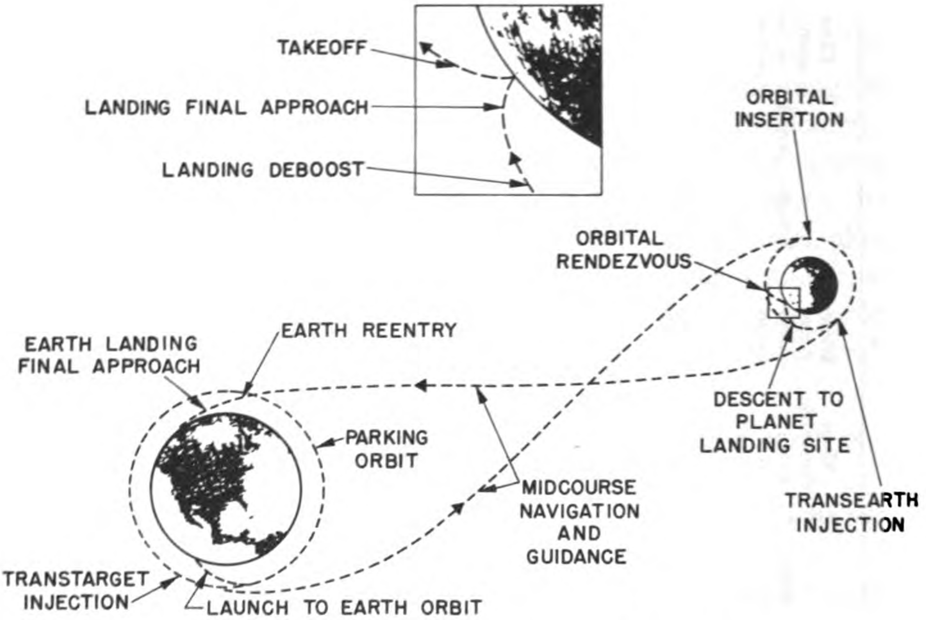

FIGURE 21-2. *Summary of Apollo mission phases* (CHAMBERLIN, 1964).

The navigation problems involved in spacecraft guidance require information concerning six initial conditions. These are the three coordinates of position and velocity (Funk, 1964). Since the errors in an inertial navigation system grow with time, this technique is not applicable over long periods. The use of angle measurements between the planets and stars provides data which are not dependent on time. The basic process involved in determining spacecraft position by means of celestial sightings consists of measuring angles between selected pairs of stars or planets. The position can be determined uniquely by using three independent and precise angular measurements made at a known instant of time. Achieving the three measurements simultaneously is impractical without extremely complicated instrumentation. However, since the vehicle dynamics are known, the problem may be linearized whenever deviations from a predetermined reference trajectory are kept small. Then the requirement for simultaneous measurements may be replaced by a series of consecutive measurements coupled with suitable computations.

The trajectories followed during space travel can be placed into three categories: ballistic, minimum energy, and constant thrust (Wall, 1958). An example of a ballistic trajectory is the first lunar probe. Lunar and Mars missions probably will use minimum energy trajectories in which only slight changes are made to the initial velocity vector. Due to the extreme distances involved, travel to the far planets and the stars will require constant thrust trajectories.

Figure 21-3 shows a minimum energy trajectory, called a Hohmann Ellipse, as it would be used during an Earth to Mars mission. The time required to reach Mars would be 283 days. Earth and Mars must initially be in proper position with respect to each other. This position is indicated by Eo and Mo. The relative positions of Earth and Mars are shown at points 1 through 4 as the voyage progresses.

Rendezvous Techniques.—The bringing together of two spacecraft in an orbital rendezvous is a delicate process involving different kinds of guidance and control problems from those found either with automobiles or with aircraft. In rendezvous maneuvers, there are two major changes which must be made in the flightpath of the spacecraft which is attempting to intercept the target vehicle. First, the orbital plane of the spacecraft must be adjusted so that it coincides with that of the target as shown in figure 21-4. This is true unless the launch is conducted with such precise timing that the initial entry into orbit is coplanar with the target. If it is necessary to change the

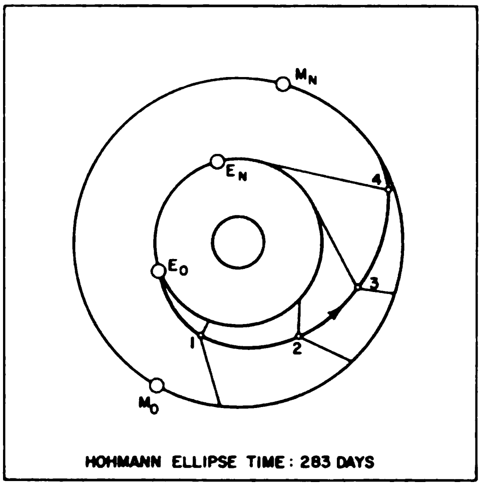

FIGURE 21-3. *Trajectory of Earth-Mars orbit* (WALL, 1958).

plane of the vehicle's orbit to make it coplanar, corrective thrust must be applied at one of the orbital nodes, which are the points where the orbit of the spacecraft intersects the plane of the target vehicle. The onboard spacecraft computer normally computes the amount, direction, and time of application of thrust necessary for a corrective maneuver. Although these values are displayed to the pilot for monitoring purposes, the maneuver normally is executed automatically.

The second necessary change in the flightpath of the spacecraft is one which places it in the same orbital path as the target. This maneuver will require a minimum of two thrust applications (Hopkins, Bauerschmidt, & Anderson, 1960). The first thrust initiates an elliptical orbit with apogee (high point) at the orbit of the target. The second thrust achieves circular orbital velocity at that altitude.

The application of thrust to achieve identical orbits must be timed so that the spacecraft enters the orbit of the target in the vicinity of the target. It is impossible for the spacecraft, once it is in the target orbit, either to "speed up" in order to overtake or "slow down" in order to be overtaken by the target (Hopkins et al., 1960). Any change in velocity of the spacecraft, either an increase or decrease from circular velocity at the altitude of the target, will change the eccentricity of its orbit. In other words, any attempt to speed up in order to overtake the target will simply result in the spacecraft being placed in a higher orbit above the target. However, if the spacecraft is in the

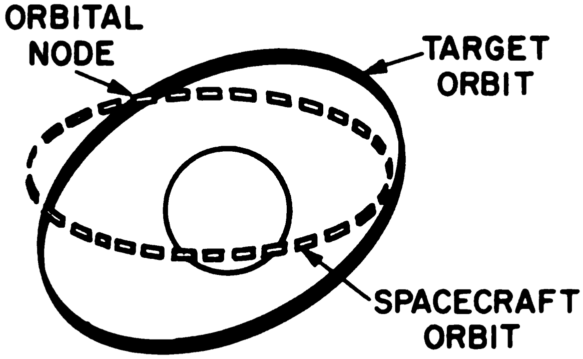

FIGURE 21–4. *Orbital paths requiring change of plane.*

near vicinity of the target at the time of injection into the target orbit, minor increases or decreases in velocity, accomplished by means of the proper combination of amount and direction of thrust applications, may be used to close on the target. This "brute force" closing maneuver is possible for short ranges in the order of several thousand feet.

Medical Sciences in Space Operations

The manned spacecraft required to traverse the tremendous distances of space is no ordinary vehicle. The needs of the man and machine are so interrelated, so mutually dependent, that they must be treated as a single system consisting of mutually complementary human and mechanical elements. The scope, as well as the success, of future manned space missions will depend upon the support and effective utilization of man for these extended periods of time. The understanding of man's performance capability and psychophysiological limitations directly affects design considerations and is essential to insure the effective integration of man as a functional part of a complete space system.

There is a great need to determine the human technological requirements for extended space flight and to develop the technology of life support for such missions. Both in civilian and military organizations, there is a growing requirement for medical personnel both to study human performance in the space environment and to provide medical support for manned space flights. Navy Flight Surgeons already are participating in the extensive life science research program being supported by the National Aeronautics and Space Administration. Much of this program is conducted by Navy facilities. The

Naval Aerospace Medical Institute, Pensacola, is studying the effect of various rotational forces on human performance and physiological functioning. The Aerospace Medical Research Department, Johnsville, is investigating human response to the high acceleration forces of launch and reentry. These are but two examples. Virtually every Navy medical research activity is engaged in some research which contributes to our national space effort.

NATIONAL AERONAUTICS AND SPACE ADMINISTRATION

The organizational structure of the National Aeronautics and Space Administration is shown in figure 21–5. The Administrator and four primary Offices comprise NASA Headquarters, located in Washington, D.C. Primary direction and coordination of space programs is accomplished at this level. The following are the primary missions of these offices:

Office of Manned Space Flight

This office is responsible for the planning and conduct of manned space flight operations, including the development of flight vehicles and associated equipment.

During actual space operations, Flight Surgeons often are assigned as medical monitors to observe telemetered biomedical information as it is received at tracking stations around the globe. These assignments may involve duty at locations as far removed as the Australia tracking station. In addition to the monitoring operation, Flight Surgeons in this assignment provide close medical support during recovery operations in the event such is needed. These medical operational and planning functions, together with related research and development activities associated with approved manned space programs, such as Mercury, Gemini, and Apollo, fall under the cognizance of the Director of Space Medicine located within the Office of Manned Space Flight.

Office of Space Science and Applications

This office is responsible for the planning and conduct of primarily unmanned space flights in a program concerned with scientific explorations of space and the detection of extraterrestrial life.

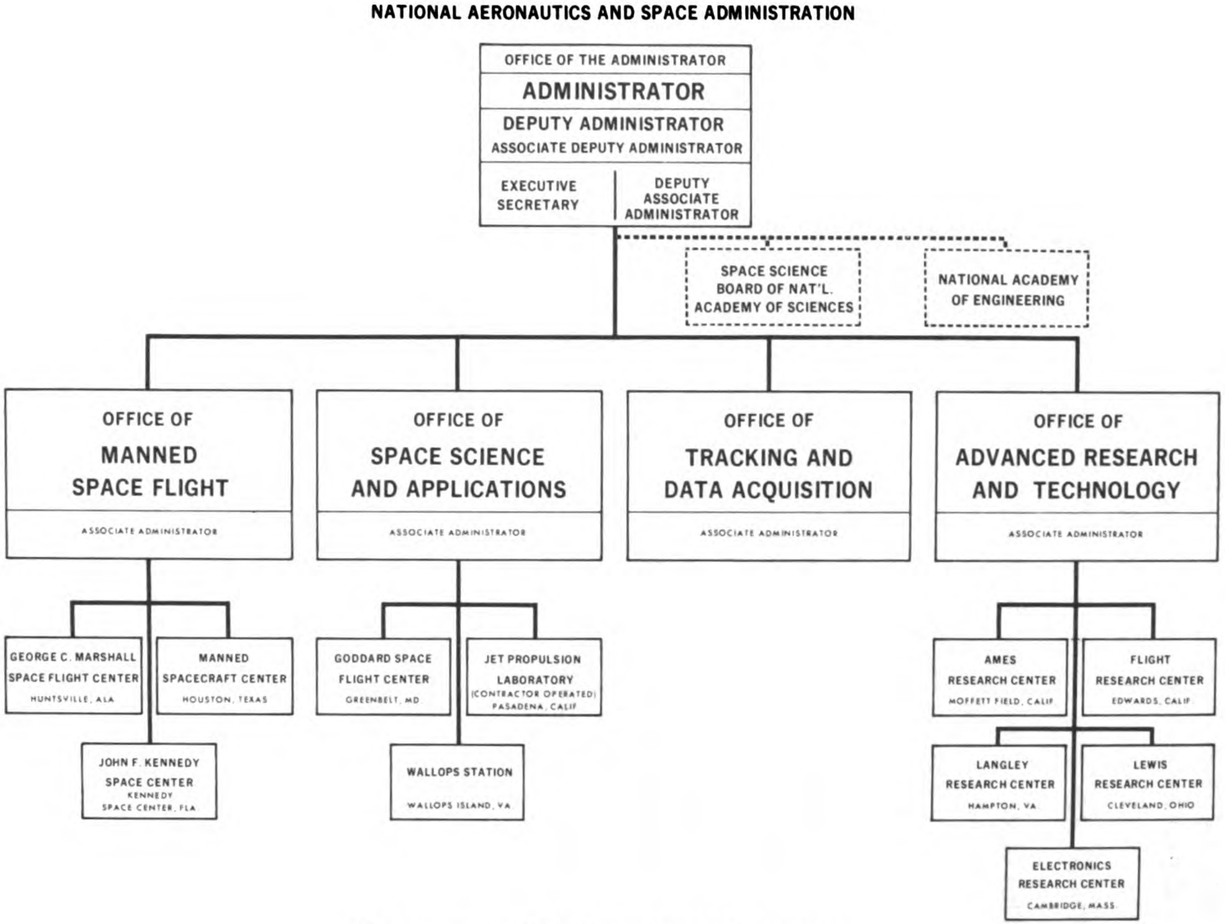
NATIONAL AERONAUTICS AND SPACE ADMINISTRATION
OFFICE OF THE ADMINISTRATOR
ADMINISTRATOR
DEPUTY ADMINISTRATOR
ASSOCIATE DEPUTY ADMINISTRATOR
EXECUTIVE SECRETARY
DEPUTY ASSOCIATE ADMINISTRATOR
SPACE SCIENCE BOARD OF NAT'L. ACADEMY OF SCIENCES
NATIONAL ACADEMY OF ENGINEERING
OFFICE OF MANNED SPACE FLIGHT
ASSOCIATE ADMINISTRATOR
OFFICE OF SPACE SCIENCE AND APPLICATIONS
ASSOCIATE ADMINISTRATOR
OFFICE OF TRACKING AND DATA ACQUISITION
ASSOCIATE ADMINISTRATOR
OFFICE OF ADVANCED RESEARCH AND TECHNOLOGY
ASSOCIATE ADMINISTRATOR
GEORGE C. MARSHALL SPACE FLIGHT CENTER
HUNTSVILLE, ALA
MANNED SPACECRAFT CENTER
HOUSTON, TEXAS
JOHN F. KENNEDY SPACE CENTER
KENNEDY SPACE CENTER, FLA
GODDARD SPACE FLIGHT CENTER
GREENBELT, MD
JET PROPULSION LABORATORY
(CONTRACTOR OPERATED)
PASADENA, CALIF
WALLOPS STATION
WALLOPS ISLAND, VA
AMES RESEARCH CENTER
MOFFETT FIELD, CALIF
FLIGHT RESEARCH CENTER
EDWARDS, CALIF
LANGLEY RESEARCH CENTER
HAMPTON, VA
LEWIS RESEARCH CENTER
CLEVELAND, OHIO
ELECTRONICS RESEARCH CENTER
CAMBRIDGE, MASS

FIGURE 21–5. *NASA organizational structure.*

Office of Tracking and Data Acquisition

This office is responsible for the planning, construction, and operation of ground instrumentation systems, including global tracking networks and data acquisition systems.

Office of Advanced Research and Technology

This office is responsible for the planning and conduct of research and technological programs concerned with advanced concepts, structures, components, and systems having possible application to the objectives of NASA. Within OART, the life sciences research program is called the Human Factors System and is directed through the Biotechnology and Human Research Division. Figure 21–6 shows the major subdivisions within this division.

Implementation of the Human Factors Systems Program is accomplished at NASA centers, located in many parts of the United States. Within these centers, most basic research in the life sciences is accomplished at the Ames Research Center, Moffett Field, California. Other NASA centers of particular interest to the Flight Surgeon are the Langley Research Center, which investigates life support systems and where versatile mission simulation

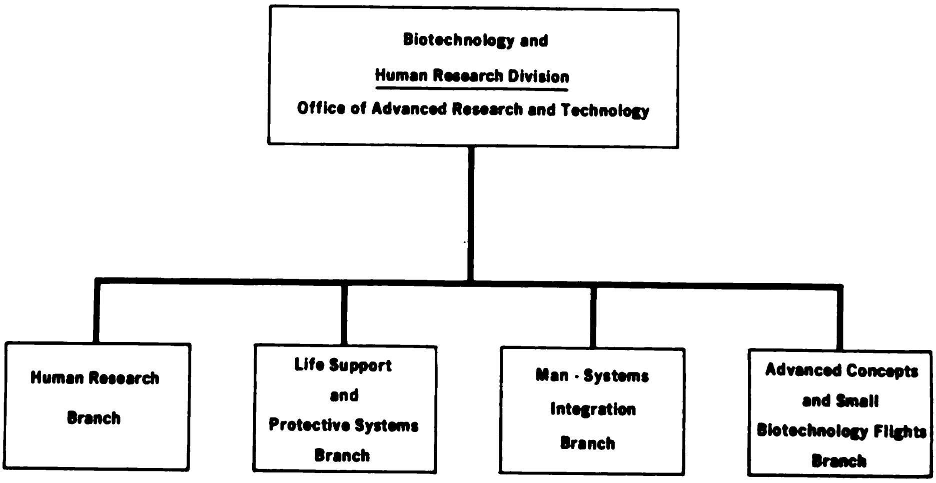
Biotechnology and
Human Research Division
Office of Advanced Research and Technology
Human Research Branch
Life Support and Protective Systems Branch
Man - Systems Integration Branch
Advanced Concepts and Small Biotechnology Flights Branch

FIGURE 21–6. *Major subdivisions in the NASA Biotechnology and Human Research Division.*

equipment is available; the Flight Research Center, Edwards, California, where research in biomedical monitoring is accomplished; the Electronics Research Center, Boston, Massachusetts, which deals with problems of space telemetry and biomedical monitoring; the Marshall Space Flight Center, Huntsville, Alabama, where human engineering problems in the use of space equipment are studied; and the Manned Spacecraft Center, Houston, Texas, where human research is conducted, protective and life support equipment for astronauts developed, and operational missions controlled.

BIOMEDICAL MONITORING

Advances in telemetry and computer sciences have made it possible for information relating to the well-being of an astronaut to be displayed and analyzed in real time. Blood pressure, pulse rate, respiration rate, body temperature, and electrocardiographic information are the variables customarily telemetered. Through a consideration of changes in each variable, as well as the pattern provided by all variables, the Flight Surgeon can judge whether an astronaut is physiologically capable of continuing a mission.

New Techniques

Part of the current Human Factors systems research program in OART is directed toward the development of improved techniques for the monitoring of physiological functioning while astronauts are involved in many different activities. Personnel at the NASA Flight Research Center are developing improved techniques for biomedical monitoring. Figure 21–7 shows electrodes which are applied to the subject's skin using a spray technique. The electrode consists of finely divided silver suspended in thinner for spray application. Figure 21–8 shows the spray gun used in electrode application. These techniques allow a subject to be completely instrumented for biomedical monitoring in less than three minutes.

The telemetry equipment developed at the Flight Research Center has been tested under combat conditions using Navy carrier pilots as subjects. It was surprising to find that heart rates were higher, as shown in figure 21–9, during the periods just prior to carrier launch and recovery than during actual bombing runs. In other studies with experienced pilot populations, risk was

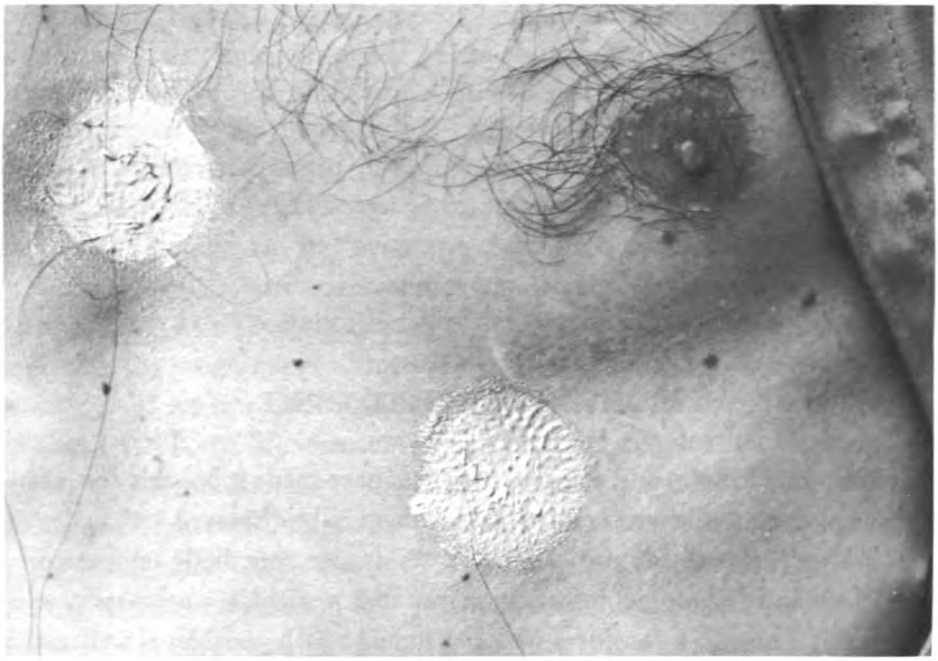

FIGURE 21–7. *Attachment of spray electrodes* (NASA PHOTO).

found to have a negligible effect upon heart rate, whereas responsibility caused large changes.

Orbital Data

Heart Rate.—Much information concerning the effects of the space environment on cardiac functioning has been obtained through data telemetered during orbital missions. In all such missions, peak elevations of heart rate have occurred in launch and reentry (Berry, Coons, Catterson, & Kelley, 1966). Table 21–2 shows peak heart rates during launch and reentry for the command pilot and the pilot of five Gemini flights. In general, there is a decrease in heart rate from an initial high level at launch toward a stable and lower baseline rate during the midportion of the mission. This is altered at intervals as the heart responds to the demands of inflight activities. The rate stabilizes around the 36- to 48-hour period and remains at this lower level until two or three revolutions before retrofire. The anticipation and activity associated with preparation for retrofire and reentry cause an increase in heart rate for the remainder of the flight.

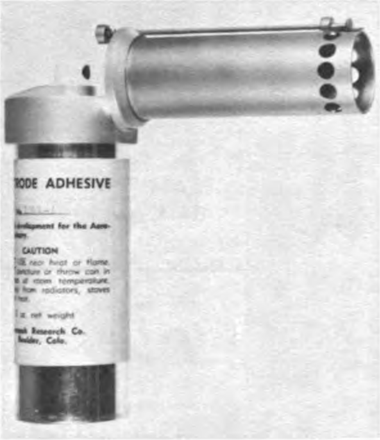

FIGURE 21–8. *Spray electrode gun* (NASA PHOTO).

Figure 21–10 shows the changes which occurred in Major Edward White's heart rate during the early hours of his flight on Gemini IV. The first sharp rise (1) occurred at liftoff at Cape Kennedy. There was a rise to about 150 beats per minute during his "walk" in space, rising to a rate of 180 per minute as he struggled to close a defective hatch on his return (2). Another peak (3) came as he performed with an elastic exerciser. These variations in heart rate clearly demonstrate that medical monitoring involves more than a simple analysis of the telemetered trace.

Blood Pressure.—The only truly remarkable thing in all blood pressures recorded during space activities to date has been the normal character of these readings (Berry et al., 1966). There has been no significant increase or decrease with prolonged space flight. Blood pressures have varied with heart rate, as evidenced by the 200/90 blood pressure obtained after retrofire during one of the missions. This was accompanied by a heart rate of 160, however, and is felt to be entirely normal for this activity.

Respiration.—Respiration rates recorded during orbital activities all have fallen within a normal range and have tended to vary with heart rate. No evidence of hyperventilation has been noted during any flight.

Orthostatic Hypotension.—The first effect of manned space flight considered to be abnormal was the postflight orthostatic hypotension observed on the last two Mercury missions (Berry et al., 1966). On four-, eight-, and fourteen-day Gemini missions, no symptoms of faintness were experienced by crewmembers at any time during the landing sequence or during post-

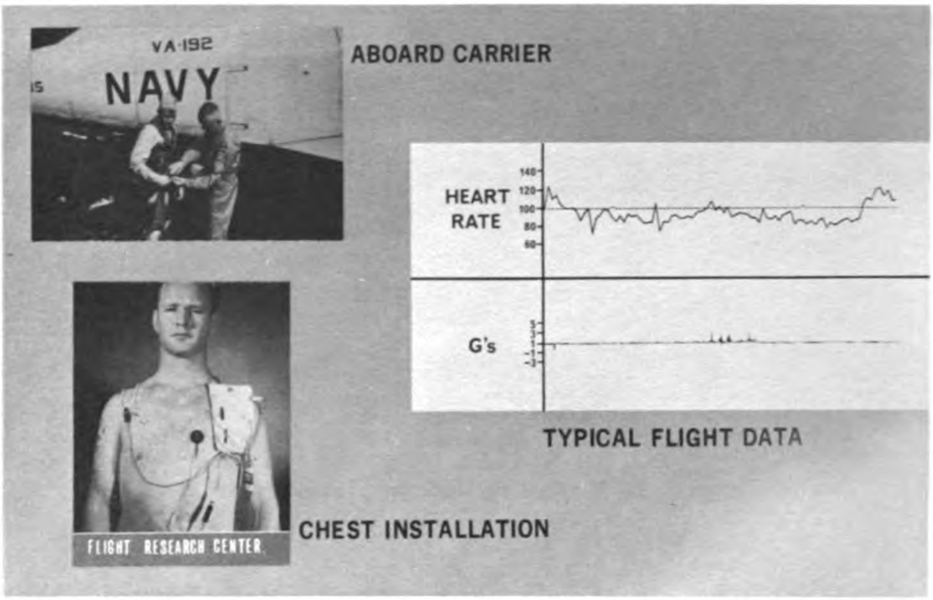

FIGURE 21–9. *Physiological data from operational aircraft flights* (NASA PHOTO).

landing operations. Abnormal postflight tilt table responses have been observed, however, when compared with the preflight baseline tilts. These have lasted for a period of 48 to 50 hours following landing. Figure 21–11 shows pre- and postflight tilt table results for the Command Pilot of Gemini V.

TABLE 21–2.—*Peak Heart Rates, Beats/Min., Recorded During Gemini Missions*

Flight	Launch	Reentry
Gemini III	[1] 152	165
	[2] 120	130
Gemini IV	148	140
	128	125
Gemini V	148	170
	155	178
Gemini VI	125	125
	150	140
Gemini VII	152	180
	125	134

[1] Command Pilot.

[2] Pilot.

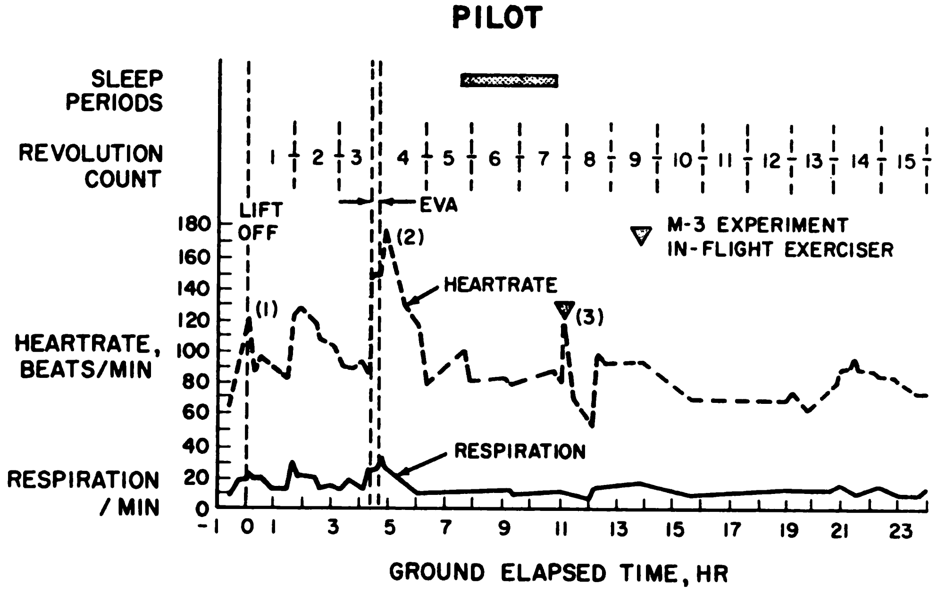

FIGURE 21–10. *Variation in heartrate during space operations* (NASA GEMINI V REVIEW CONFERENCE, 1965).

A comparison of results for missions of different duration has shown no increase in the time required for an individual to return to the normal preflight tilt response. In all instances, approximately 50 hours are required to readjust to the one-G environment. Strain gauge data generally confirm pooling of the blood in the lower extremities during this period.

Disorientation.—Although early space flights have not given rise to any disorientation effects on the part of astronauts (Berry et al., 1966), the potential for such effects during long space flights is such that NASA is supporting an intensive investigation of the primary human balance mechanisms. The center of this research is in the Naval Aerospace Medical Institute under the direction of Dr. Ashton Graybiel.

Since the normal condition of balance exists in a one-G environment, the effects of removing this gravitational framework must be determined. If it is found that man cannot adapt to long-term weightlessness, it may be necessary to substitute an artificial gravity such as would be produced by rotation of the spacecraft. If this is necessary, the ideal rates of rotation must be determined. To acquire this information, a number of manned rotating tests are being conducted, using varying speeds and rates of onset.

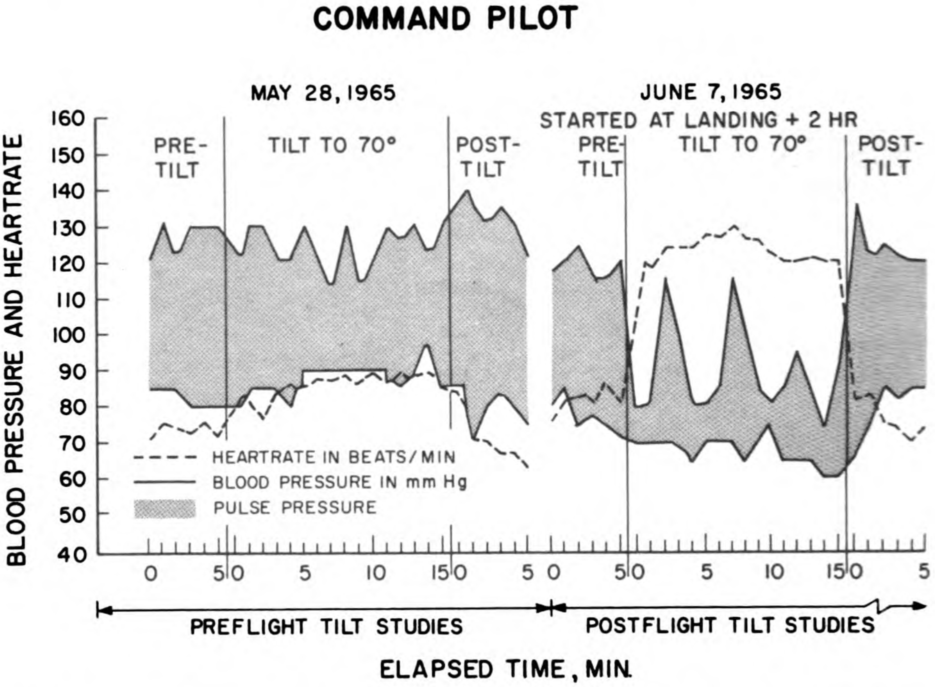

FIGURE 21-11. *Changes in blood pressure and heartrate during pre- and postflight tilt table studies* (NASA GEMINI V REVIEW CONFERENCE, 1965).

In other tests of the adaptation of the vestibular system to different gravitational effects, Dr. Graybiel is studying ocular counterrolling: the compensatory or counterrotation of the eye when the head is tilted to one side (Miller & Graybiel, 1962). "Counterrolling" is a fast-acting reflex response. Since man cannot voluntarily roll his eyes in the frontal plane, this response is distinguishable from all voluntary eye movements. Although the counterrolling phenomenon had been identified for a long time, this reflex has received little attention because of the difficulties in obtaining precise measurements. The measurement problems are now being solved, however. Using the counterrolling test, data have been obtained comparing normal subjects and those with vestibular (otolith) defects. While the eyes of the normal man counterroll five to six degrees on the ground, the eyes of vestibular-defective subjects roll little or none. Results of these tests are providing basic information concerning likely adaptation processes related to altered gravitational exposures which will be found in space.

UNIQUE OPERATIONS AND PROBLEMS IN THE SPACE ENVIRONMENT

Human performance in the space environment poses a number of medical problems which arise both from the different operating conditions, such as altered work-rest cycles, and from unusual stress agents, such as increased radiation levels. For certain of these topics, it is possible only to discuss the problem itself. Answers to the problem must await information obtained either from extended space flight or from the definitive research. For other problem areas, sufficient information has been accumulated that meaningful predictions can be made as to man's response to this problem area and personal protective equipment developed on the basis of these predictions.

The following sections describe some of the principal problem areas to be dealt with as preparations are made for extended space flight. Certain critical problems, such as the provision of oxygen, are not discussed since they have been covered in earlier sections of this manual. These problems are no different in space than in high-altitude flight in excess of 50,000 feet. The same technology provides solutions in either instance.

Weightlessness

Weightlessness has been considered as one of the major problems to be overcome in preparing man for space flight. This apprehension was due to the truly unique quality of this experience and the considerable difficulty in simulating this stress condition within the earth environment. In none of man's everyday experiences is he in a weightless state. This means that none of the organs within the human body have had an opportunity to accomplish any measure of adaptation to this condition. Following thirty some years of exposure to a one-G environment, it is reasonable to expect that the body might react rather strangely in the complete absence of this force.

Simulation of weightlessness has proved quite difficult. Aircraft, flying what is known as a zero-G or Keplerian trajectory, have been able to achieve periods of weightlessness of up to one minute. For the larger aircraft, in which experiments can be carried out, the period of induced weightlessness ranges from 15 to 25 seconds. However, even this is sufficient for the practice of certain movements and manipulative activities. The usefulness of these ded periods of weightlessness for a study of the response of specific body systems is quite restricted. The period of weightlessness generally is preceded and followed by periods of increased weight which may be even further complicated by radial accelerations. It is difficult to separate effects due to weightlessness from those which might be produced by the increased acceleration forces.

Water immersion has been used as one means of simulating weightlessness for extended periods with the subject weighted to the point of neutral buoyancy. However, different forces still act on and displace internal organs. For this reason, and because the subject is immobilized during the entire period of immersion, this procedure is limited to simulating extravehicular activities for long periods of time. Water immersion is felt to be of little value in assessing bodily changes due to weightlessness.

Fortunately, the results of actual space missions to date indicate weightlessness to be much less of a problem than had been initially feared. In fact, it seems to provide a somewhat exhilarating experience for astronauts and may even provide certain benefits in terms of reducing the fatigue which would be felt when remaining in the seated position for long periods, if one were in a one-G environment.

In reports of one experience, which may be related to weightlessness, crews have described a sensation of fullness in the head that occurred during the first 24 hours of the mission and then gradually disappeared (Berry et al., 1966). This feeling is similar to the increase of blood a person notes when hanging inverted on parallel bars or when standing on his head. There was no pulsatile sensation in the head and no obvious reddening of the skin. The exact cause of this condition is unknown, but it may be related to an increase of blood in the chest area as a result of the readjustment of the circulation to the weightless state.

Although initial reports of the effects of weightlessness have been encouraging, potential problem areas remain. For instance, X-ray density measurements before and after the four-day flight of Gemini IV showed about a ten percent drop in bone density. The extent to which this calcium loss can be arrested through inflight exercise procedures is not known precisely at this time, although results of the 14-day Gemini VII mission, in which an exerciser was used, show a smaller decrease in bone density. However, this may be the result of an overall improvement in flight conditions, including better rest and food combined with exercise, over that of earlier flights.

Extravehicular Activities

One of the most unique operating conditions for an astronaut is that of extravehicular activity (EVA), that period during which he must operate without the shelter and protection of a spacecraft. This means that all life support requirements, such as those for oxygen and pressurization, must be met by the astronaut's garments or by a direct attachment to the spacecraft. There is also the problem of providing adequate radiation shielding without unduly increasing the bulk of the protective clothing. The most important problem, however, seems to be that of seeing that the astronaut can actually perform those tasks required of him during periods of EVA. Studies to identify these extravehicular activities and to determine the most effective methods for their accomplishment have provided the following categories:

1. Maintenance and repair of own and other vehicles and satellites.
2. Transfer of personnel and cargo.
3. Assembly and erection of objects.
4. Refurbishing and resupply of vehicles and satellites.
5. Guidance and assistance to docking and similar maneuvers.
6. Personnel rescue.
7. Retrieval of objects.
8. Performance of scientific tasks.

For determining specific maintenance, repair, and assembly tasks, the Marshall Space Flight Center has a program to study the tools and tasks required for assembly and checkout of boosters and for launch preparations from orbit. As part of this program, the effectiveness of standard tools during weightlessness is being investigated.

The Langley Research Center has an intensive effort underway to study systems which might assist an astronaut during extravehicular activities. The use of integrated personnel propulsion devices which incorporate integral life support and environmental control units is being studied, using the suspension simulation facilities at Langley. Figure 21–12 shows a man in such a unit suspended from the same carriage normally used for the Agena docking simulator. Of particular interest in these studies is the ability of such a maneuvering device to assist an astronaut in moving large masses under low relative velocity conditions.

A second personnel propulsion device under investigation at Langley utilizes jets of compressed gas attached to the shoe soles in such a manner as

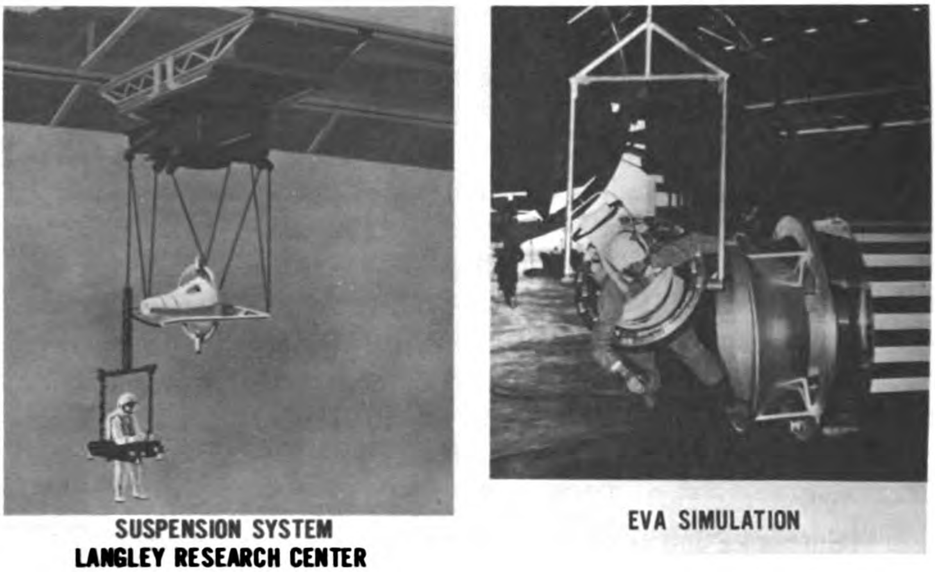

FIGURE 21–12. *Personnel propulsion maneuvering device* (NASA PHOTO).

to produce a thrust vector along the leg when activated by a switch under the toes. Figure 21–13 illustrates the operation of these shoes. The jets of each shoe are independently controlled. Use of devices such as the jet shoes should do much to alleviate the heavy physical exertion normally experienced during extravehicular activities.

Radiation Environment

The radiation environment in space, differing substantially from that found on earth, represents a hazard which under certain conditions could place a limiting constraint on space travel. For this reason, the National Aeronautics and Space Administration has a substantial program to define this environment and to develop a complete understanding of the biological effects of exposure to increased radiation levels.

Units of Measure.—In describing radiation and its effects on man, several units are used, which are interrelated as follows (*Nuclear Radiation Guide,* 1962):

Roentgen: The roentgen is a basic indicator of quantity of radiation. Inasmuch as radiation cannot be measured directly, its magnitude is deter-

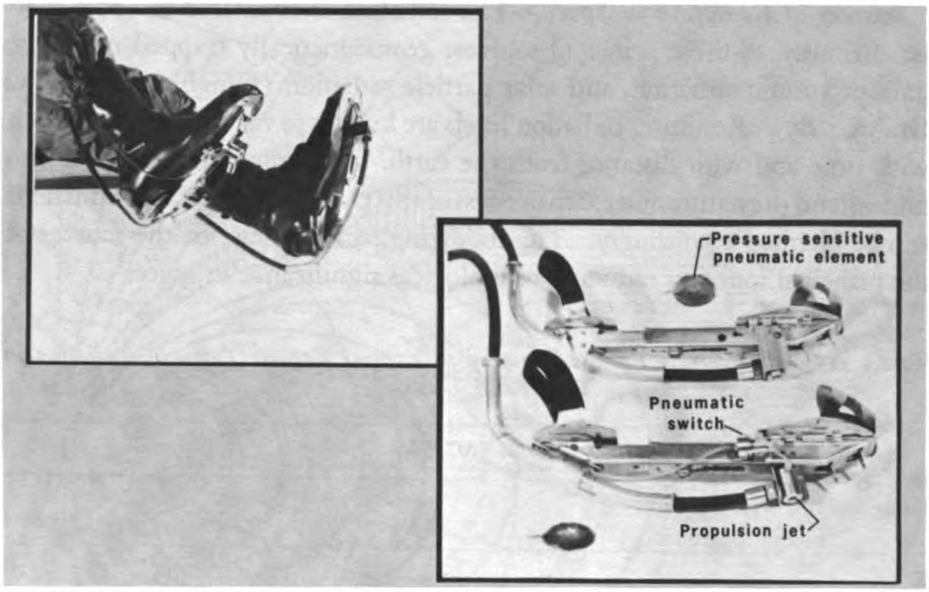

FIGURE 21–13. *Jet shoes propulsion device* (NASA PHOTO).

mined by the ionization produced by the passage of radiation through a medium. The roentgen refers to the ionization produced in air by the passage of X or gamma radiation, specifically, that amount of radiation required to produce 0.001293 grams of air ions carrying one electrostatic unit of electricity of either sign.

Rad: The rad (radiation absorbed dose) is a more universally used measure of exposure. One rad of any type of radiation corresponds to the absorption of 100 ergs per gram of any medium. Because the rad does not specify the medium, a medium should be stated unless clearly implied. For example, the term "tissue rad" should be used in the case of exposure of soft human tissue.

Rem: The rem (roentgen equivalent, man) is the most accurate unit for expressing exposure of man. The rem refers to the absorbed dose of any ionizing radiation which produces the same biological effects in man as those resulting from the absorption of one roentgen of X-rays. Each type of radiation must be converted by a specific factor, the RBE (relative biological effectiveness), in order to equate the biological effects. Table 21–3 presents RBE factors for different types of radiation. A dose expressed in rads may be converted to rems by multiplying the rad value by the RBE factor.

Sources of Radiation in Space.—The radiation encountered in space may be attributed to three principal sources: geomagnetically trapped radiation, galactic cosmic radiation, and solar particle radiation (Langham, Brooks, & Grahn, 1965). Resulting radiation levels are known to vary substantially both with time and with distance from the earth. It is important, therefore, to understand the nature and extensiveness of this radiation so that space missions can be planned accordingly. The following sections describe the sources of the principal ionizing radiation of biological significance in space.

TABLE 21–3.—*Conversion Factors Required to Express Various Types of Radiation in REM Units*

RBE FACTORS

Energy and type of radiation	RBE
X	1
Gamma	1
1.0-Mev* Beta	1
0.1-Mev Beta Particle	1.08
Thermal Neutron (Mev)	2 to 5
1.0-Mev Proton	8.5
0.1-Mev Proton	10
Fast Neutron (1 to 10 Mev)	10
5-Mev Alpha Particle	15
1-Mev Alpha Particle	20

*Mev = million electron volts.

(*Nuclear Radiation Guide*, 1962.)

1. *Geomagnetically Trapped Radiation.* Small amounts of radiation from space, when approaching the earth, can be trapped in its magnetic field by being forced into closed trajectories within rather well-defined boundaries. Within these radiation belts, the buildup of radiation reaches such proportions that the resulting intensities make these regions uninhabitable for man without adequate shielding, even for an exposure of only several hours.

There are two belts of geomagnetically trapped radiation around the earth. These belts, known as the Van Allen belts, are separated by a region of relatively low intensity, as shown in figure 21–14. In the equatorial plane, the inner Van Allen belt begins at an altitude of about 600 km (370 miles) and extends to about 10,000 km (6,200 miles). Maximum intensity is found at an altitude of around 4,000 km (2,500 miles). Radiation within the inner Van Allen belt is composed of high-energy protons and soft electrons. Pro-

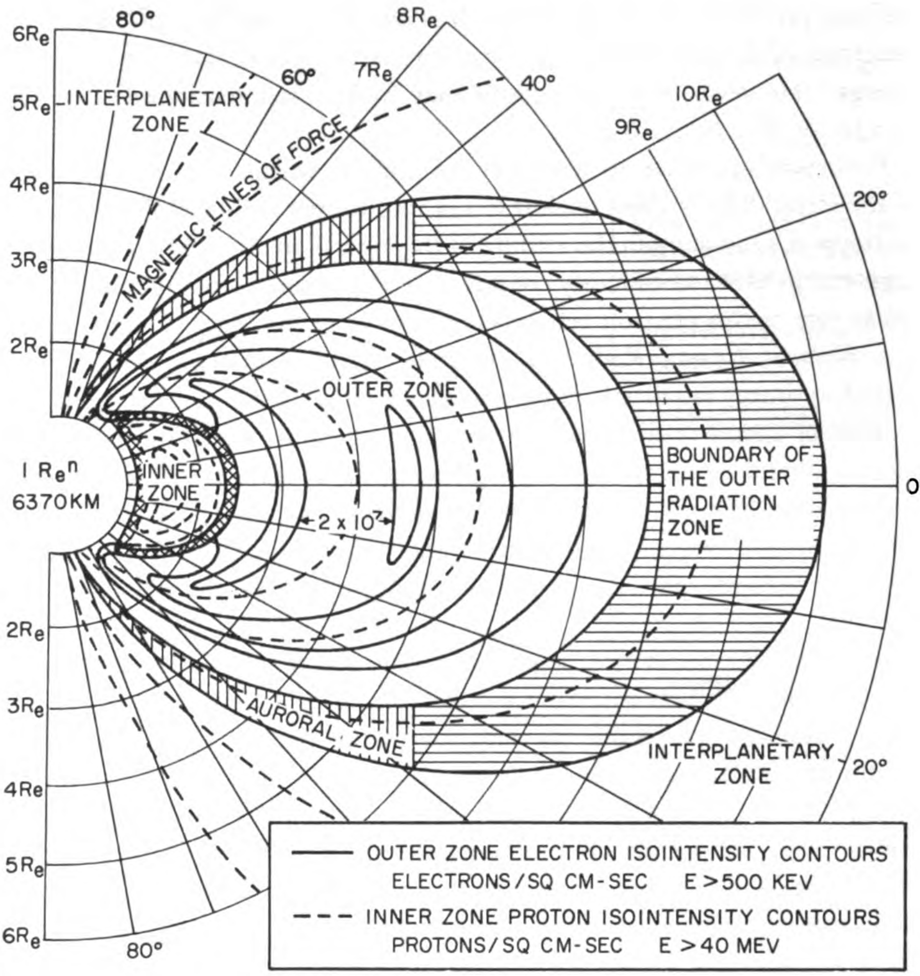

FIGURE 21–14. *Radiation belt boundaries.* (FROM "SPACE AND ASTRONOMY"BY C. D. GRAVES. IN System Engineering Handbook, R. E. MACHOL (ED.), COPYRIGHT 1965, MCGRAW-HILL. USED BY PERMISSION OF MCGRAW-HILL BOOK COMPANY.)

ton intensity, which is of primary concern in this region, achieves a maximum flux of about 20,000 protons/cm^2/sec of 40-Mev minimum energy.

At a distance of 15,000 km (9,000 miles) from the earth, radiation intensity is at a minimum. As distance increases, intensity levels again rise and reach a second maximum at approximately 20,000 km (12,000 miles). This outer Van Allen belt extends out to 70,000 km (44,000 miles). Primary particles within the outer belt are electrons ranging from about 50 Kev to a few Mev and soft protons with energy of less than one Mev. Intensity of electrons at the center of this belt is about 20,000 electrons/cm^2/sec of 500-Kev minimum energy. However, magnetic storms may cause this intensity value to vary by a factor of as much as 20.

Fission debris electrons also may be trapped within the magnetic field of the earth. The Starfish nuclear test in 1962 caused high-energy electrons to be trapped, producing in the vicinity of the inner Van Allen belt a maximum high-energy electron intensity considerably above that normally found. The decay rate of this electron belt is believed to be in the order of years.

2. *Galactic Cosmic Radiation.* Galactic cosmic radiation, frequently referred to simply as galactic radiation, originates outside the solar system and consists of atomic nuclei which have been completely ionized (stripped of all electrons) and accelerated to very high energies. Protons of 10^{18} electron-volt energy have been identified from this source. Protons (hydrogen nuclei) constitute about 85 percent of this irradiation; alpha particles (helium nuclei), about 13 percent; and heavier nuclei ranging up to tin, the remaining few percent.

Measures obtained with deep-probe rockets indicate a fairly constant cosmic ray intensity between 1.0 and 1.5 astronomical units from the sun. This would indicate that cosmic ray intensity in the vicinity of planets such as Venus and Mars is quite similar to that found near the earth.

3. *Solar Particle Radiation.* The greatest radiation hazard for space flight comes from solar particle beams emitted during solar flares. During a period of increased solar activity, high-energy particles are radiated throughout the solar system. These particles are mostly protons, with a lesser quantity of alpha particles and higher atomic number nuclei.

Solar flares are relatively rare events, occurring only a few times during the period of high activity in the 11-year solar cycle. Flares develop rapidly and typically last only 30 to 50 minutes. However, the increased proton radiation continues for a period of up to 36 or even 48 hours after the flare.

Solar flare radiations are hazardous since it is quite difficult to predict sufficiently in advance the size and time of occurrence of a specific event. In addition, proton flux may increase by several orders of magnitude.

Radiation Damage.—The primary event associated with radiation damage is energy transfer and is essentially the same in all cells and cell systems (Fox, 1966). Figure 21–15 delineates the events in radiation damage schematically. The mechanism of action of this physical perturbation, like any other physiological change or stimulus, involves the usual interplay of factors such as

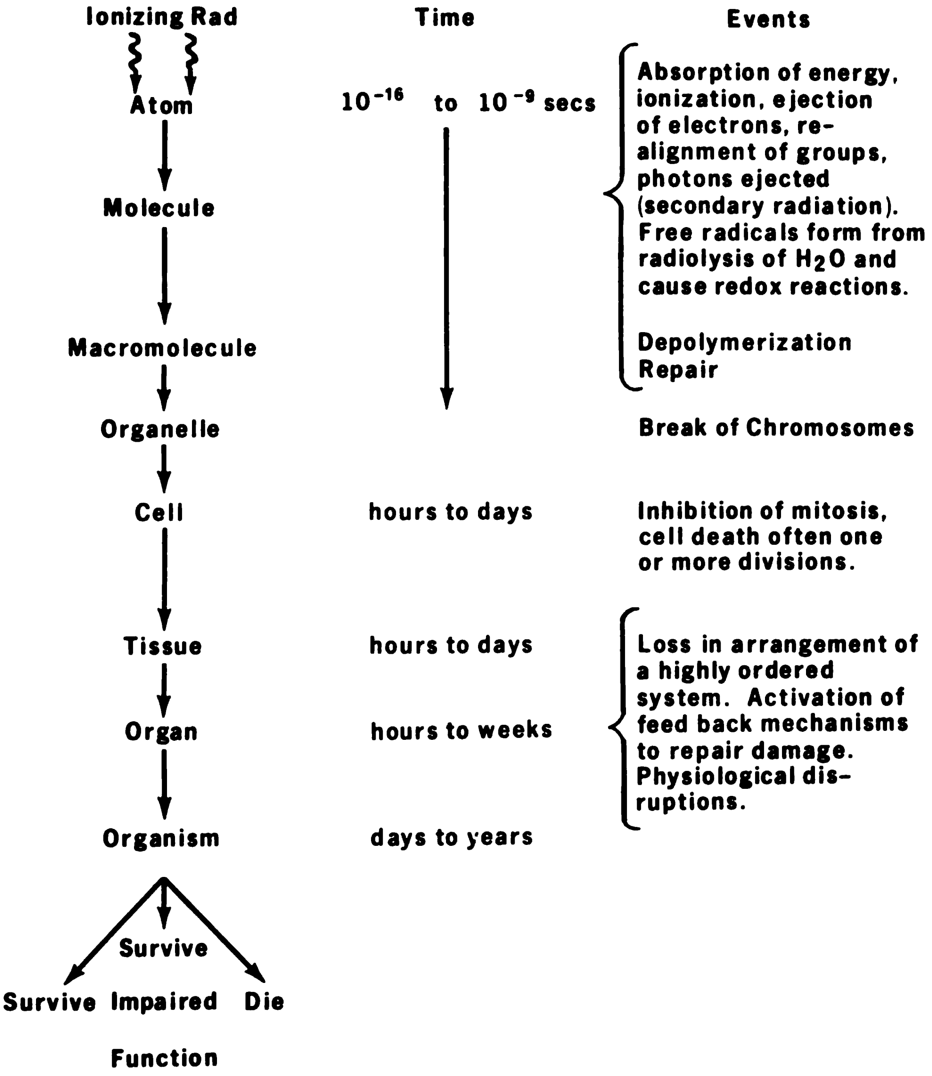
Ionizing Rad
Time
Events
Atom
10⁻¹⁶ to 10⁻⁹ secs
Absorption of energy, ionization, ejection of electrons, re-alignment of groups, photons ejected (secondary radiation). Free radicals form from radiolysis of H₂O and cause redox reactions.
Molecule
Macromolecule
Depolymerization
Repair
Organelle
Break of Chromosomes
Cell
hours to days
Inhibition of mitosis, cell death often one or more divisions.
Tissue
hours to days
Loss in arrangement of a highly ordered system. Activation of feed back mechanisms to repair damage. Physiological dis-ruptions.
Organ
hours to weeks
Organism
days to years
Survive
Survive Impaired Function
Die

FIGURE 21–15. *Schematic representation of radiation effects* (ADAPTED FROM BACQ & ALEXANDER, 1961).

threshold, intensity duration, summation, adaptation, and recovery (Baker, 1963). The concept of threshold is particularly bothersome in radiobiological effects since there is no amount of radiation which has no effect, but threshold can be interpreted simply as that point at which a recognizable response is produced. The obvious consequences of any absorbed dose will be seen when a sufficient number of units have been damaged either permanently or temporarily. The difficulty here lies in the fact that all biological systems utilize the principle of redundancy both at the cellular and tissue-organ-organism level. In normal adult tissues, there exists a so-called steady state, particularly in the proliferative tissues (skin, bone, gonads, hematopoietic tissues, hair, lining of the gastrointestinal tract, etc.). In such tissues, each has a characteristic turnover time which may vary from minutes to years, and it is the kinetics of each cell system which plays a large part in the time after radiation at which cell damage becomes apparent. There is, unfortunately, little quantitative data on the dynamics of cell turnover even in the normal so-called steady state.

Radiation Exposure.—Any increase in exposure to radiation over that absorbed from natural sources, about 100 millirads/year, is considered harmful. However, a considerable increase is required in order to produce early death. Various organizations have estimated the amount of radiation to produce death in 50 percent of exposed humans within 60 days to range from about 250 to 700 rads (Langham et al., 1965). However, evidence indicates that continuous exposure to dose rates as low as 0.1 to one rad/day results in an actuarial life shortening in man of from about one to two days per rad.

Many attempts have been made to predict the exposure of astronauts to radiation during different missions. Such calculations are quite difficult to accomplish with any precision because of the large number of variables operating to complicate the picture. Since much of the radiation consists of high-energy particles, a strong buildup phenomenon develops when the radiation strikes a compact absorber. Thus tissue dose in the human body could be higher than that recorded external to the vehicle. In addition, there will be exposure from secondary radiations caused by primary particles striking the materials of the spacecraft. Secondary radiation components of most consequence are neutrons and protons produced by solar flare and geomagnetically trapped protons and the Bremsstrahlung (X-rays) produced by high-energy electrons in the natural and artificial electron belt. The Bremsstrahlung effect is quite serious since the penetrating power of these X-rays is about 1,000 times greater than that of the electrons producing them.

Measurements have been made of radiation dosage during orbital missions. Table 21-4 shows the results obtained during the 14-day Gemini VII mission. These results confirm that flight crews are exposed to very low radiation dose levels at orbital altitudes. The body dosimeters on these missions have recorded only millirad doses which are at an insignificant level (Richmond, Davis, & Lill, 1966).

Table 21-4.—*Radiation Dosage (Millirad) on 14-Day Gemini VII Mission*

Area	Command Pilot	Pilot
Left Chest	113±13.6	231± 9.0
Right Chest	192∓10.8	105∓10.5
Thigh	178∓ 4.5	163± 8.2

For comparison purposes, table 21–5 presents radiation dose levels recorded on six Russian orbital flights. Results are in reasonable agreement with those found during Gemini missions.

Table 21-5.—*Radiation Dose Levels Recorded on Six Russian Orbital Flights*

Astronaut	Duration Flight (hours)	Types of Dosimeter					
		ILK (3.2 mm Al)		IKS (0.6 mm Su)		R-3	
		Dose (mrad)	mrad/ days	Dose (mrad)	mrad/ days	Dose (mrad)	mrad/ days
Gagarin, Yu. A	1.5	0.5	8.4	....	....	....	....
Titov, G. S	25	13±2	12±2	....	....	....	....
Nikolayev, A. G	94	64±1	16±1	58±7	15±2	43±1	11±1
Popovich, P. R	71	48±1	16±1	51±7	17±2	32±1	11±1
Bykovskiy, V. F	119	75±2	15±1	81±6	16±1	50±1	10±1
Tereshkova, V. V	71	48±1	16±1	42±2	14±1	30±1	10±1

(Volynkin, 1964.)

Schaefer (1966) describes radiation levels resulting from a solar flare which occurred during a period of solar minimum. These measures were obtained during earth orbital flight. Figure 21–16 presents the profile of radiation exposure during this event. The integral flare dose for tissue surface behind 0.1 g/cm^2 shielding equals 8.3 rads over the 44-hour period of enhanced flux. The penetrating power for this radiation also was found to be low, being considerably smaller than that encountered in conventional X-ray therapy. These results indicate that the flare event would not have constituted a major hazard for a manned space mission in progress but might be a matter of concern for systems of very low shielding such as the Lunar Module or under conditions of extravehicular activity.

The shielding afforded by a spacecraft offers some protection against radiation exposure, even though secondary radiation may complicate the exposure picture. Figure 21–17 shows anticipated exposure in the inner Van Allen belt with increasing thickness of aluminum shield. Note that although the shielding provides a decrease in exposure to primary protons, there is an increase, although at a lower level, in exposure to secondary protons and neutrons.

Calculation of the protection afforded by the shielding of a spacecraft is difficult due to variations in the shielding effect. In the Apollo vehicle, shielding ranges from 1.75 g/cm^2 to 212 g/cm^2, with the higher shielding provided by such items as the fuel tanks, rocket engine, and other heavy equipment in the service module. Prediction of dose distribution through the human body in such actual systems becomes quite complex.

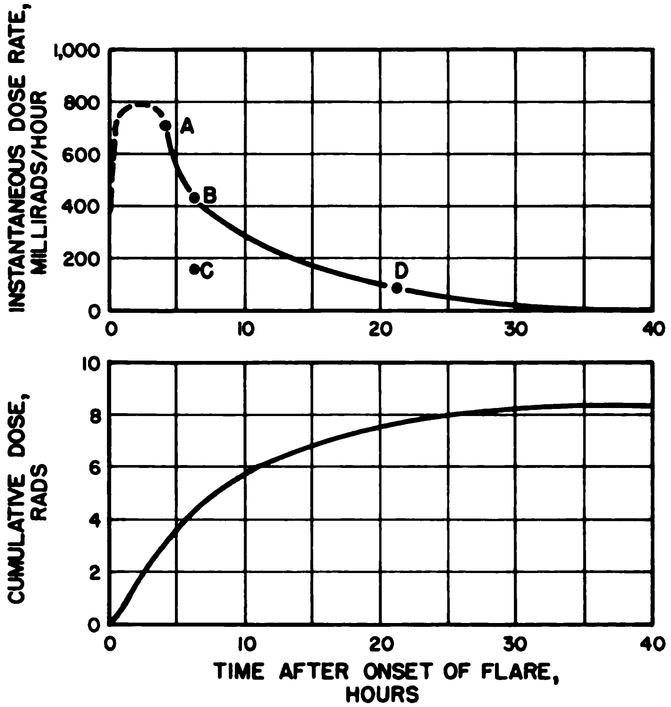

FIGURE 21–16. *Conception of time profile of radiation exposure for surface tissue behind 0.1 g/cm^2 shielding—February 5, 1965, flare* (SCHAEFER, 1966).

Oxygen Toxicity

An excess of oxygen, particularly if delivered at high pressures, is toxic. When oxygen pressures above 3.5 atmospheres are breathed, death in experimental animals may occur in two or more hours (Gell, 1961). The hyperbaric effects of an increased oxygen supply are well known and are carefully taken into account in Navy diving procedures and in submarine medicine. The effects of an increased oxygen supply at pressures less than one atmosphere are not so well established, although it is known that these effects are a function of both the excess of oxygen partial pressure in the inspired air and the duration of exposure.

For a number of years, aviators have been breathing 100 percent oxygen on flights lasting for several hours with no apparent ill effects, although there are a few reports of atelectasis in these individuals (Levy, Jaeger, Stone, & Duona, 1962). Initial orbital space flights have used a pure oxygen supply delivered at a pressure of five psi with no major changes observed which could be directly attributed to oxygen toxicity. The problem thus becomes one of determining whether there are any effects as a result of long-term exposures, in the order of thirty days or more, and if so, the maximum partial pressure of oxygen which may be used to minimize these effects. There remains,

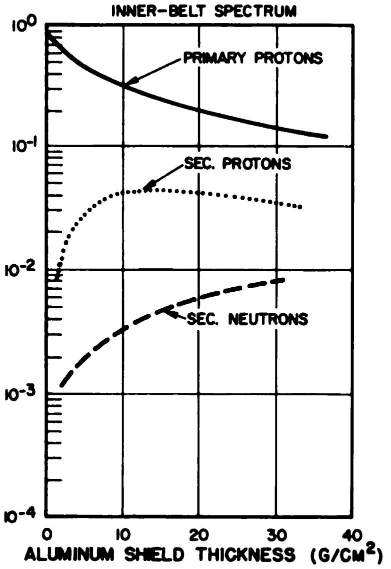

FIGURE 21–17. *Calculated primary and secondary point-target dose components as a function of spherical aluminum shield thickness* (LANGHAM ET AL., 1965).

of course, the problem of whether a one- or a two-gas environment will be needed for extended space flight. There is evidence (Nobrega, Turaids, & Gallagher, 1965) that some percentage of an inert gas such as nitrogen may be necessary in order to prevent any collapsing of the alveoli and a consequent atelectasis condition. Research results indicate that as little as five percent nitrogen in the gaseous mixture may be effective.

Damage to Pulmonary Structures.—Use of 100 percent oxygen, regardless of the pressure, may produce some symptoms in the upper respiratory tract. The drying out of the mucosa of the mouth and throat is a frequent complaint of flying personnel following prolonged breathing of 100 percent oxygen (Gell, 1961). Comroe, Dripps, Dumke, and Deming (1945) found that subjects who breathed pure oxygen at one atmosphere for 24 hours felt substernal pain and experienced a number of other symptoms of respiratory distress, such as nasal congestion, sore throat, and intermittent cough. In all, while no lung changes could be detected on X-ray, the increased oxygen supply obviously was acting as a severe irritant.

Pulmonary changes were noted, however, in a study by DuBois, Turaids, Nobrega, and Mammen (1965) in which seven subjects were exposed to 100 percent oxygen at 5 psi, 7.5 psi, and 14.7 psi for periods ranging up to 72 hours. Four of the seven subjects developed a decrease in vital capacity, with two showing a platelike atelectasis by X-ray. Of these two, one next was exposed to oxygen with 30, 5, and 2.5 percent nitrogen at 5 psi. The first two mixtures prevented the X-ray and vital capacity changes from occurring, whereas the third did not. It was concluded that the addition of nitrogen, in amounts greater than 5 percent, was sufficient to prevent the collapsing of alveoli as a result of excess oxygen absorption.

Helvey, Albright, Benjamin, Gall, Peters, and Rind (1962) also conducted a study in which one condition duplicated the spacecraft atmosphere of 100 percent oxygen at five psi. Exposure period was 14 days. Eye irritation was experienced in four of the six subjects on the second to sixth day. Mild substernal discomfort was experienced by two subjects. While there obviously are some effects of the altered oxygen supply, the general picture is improved over that found with pure oxygen at sea level pressure.

Changes in Formed Elements of the Blood.—Results in animal studies frequently have found a depressed hemoglobin count and a slightly lower reticulocyte count at oxygen pressures between 150 and 450mm Hg (Campbell, 1927). Evidence thus exists that inspiration of pure oxygen for an extended period may produce certain changes in the formed elements of the blood.

In the study of Helvey et al., using human subjects, the five-psi group "(except Subject 35) demonstrated a slight anemia, microcytosis, increased osmotic fragility, and minimal erythroid hyperactivity. Subject 37 had a loss of over 2.0 gm percent hemoglobin, and a 2.2 percent reticulocyte count. The followup examinations, nine and eleven weeks postrun, more clearly demonstrated that the hematological abnormalities of the subject had persisted." While there is some suspicion that these results may be at least in part due to cabin contaminants (Roth, 1964), they at least suggested that the pure oxygen supply may be producing certain changes, particularly in the red blood cell system.

Gemini postflight examinations have indicated some depression in red cell mass. Results of the 14-day Gemini VII flight showed no change in blood volume, 4 and 15 percent increases in plasma volume, and 7 and 19 percent decreases in red cell mass for the two crewmembers. In addition, red cell survival time was reduced. There is, of course, the problem of attributing these changes to the extended inactivity, the prolonged weightlessness, the five-psi pure oxygen supply, or a combination of the three. There remains, in addition, the question of the functional significance of this change in red cell mass.

The possibility that long-term exposures to pure oxygen may produce subtle changes in the blood picture is of concern to NASA. However, it is quite difficult to obtain data concerning this question through use of human subjects if test periods exceed 30 days. For this reason, the Ames Research Center has conducted studies, using rats as subjects, designed to examine hematological changes produced by prolonged exposure to a P_{O_2} slightly below the level where acute damage is reported to occur. Rats were maintained in a pure oxygen environment at a total pressure of 450mm Hg for 64 days (Brooksby, Dennis, & Staley, 1966). Facilities were developed for this study, as shown in figure 21–18, which allowed animal chambers to be changed and cleaned daily without exposing the animals to sea level pressure during the period of cleaning. No remarkable changes in the blood variables were noted at the conclusion of this experiment. There was a slight decrease in the red blood cell count and in hemoglobin content, attributed to the decreased RBC. These changes were considered to be the result of a minor adaptive mechanism reflecting the lengthy exposure to an increased oxygen pressure.

In a separate part of the above Ames investigation, it was found that rats exposed to a 760mm Hg pure oxygen environment for only seven days developed a lung condition similar to emphysema in man, as shown in figure 21–18. This technique may be useful in the study of emphysema since previous efforts to produce it in laboratory animals have failed.

Summary.—In general, it is felt that the effects of oxygen become toxic, for extended exposures, above a critical pressure of about 425mm Hg. Current thinking with respect to oxygen toxicity is reflected in figure 21–19, which shows the 425mm Hg limit for 100 percent oxygen and also indicates that at sea level pressure, the toxic effects do not show until the percentage of oxygen increases to over 60 percent. However, this figure does not show deleterious effects which might arise in extended exposures. Additional research and operational experience must provide answers to this question.

The space cabin atmosphere of five psi (258mm Hg) of 100 percent oxygen falls well within the safe zone shown in figure 21–19. In addition, current NASA thinking is that a two-gas environment may be provided for the longer missions of 45 days or over. In this case, the partial pressure of oxygen can be maintained at an approximately normal value and possible problems of oxygen toxicity eliminated.

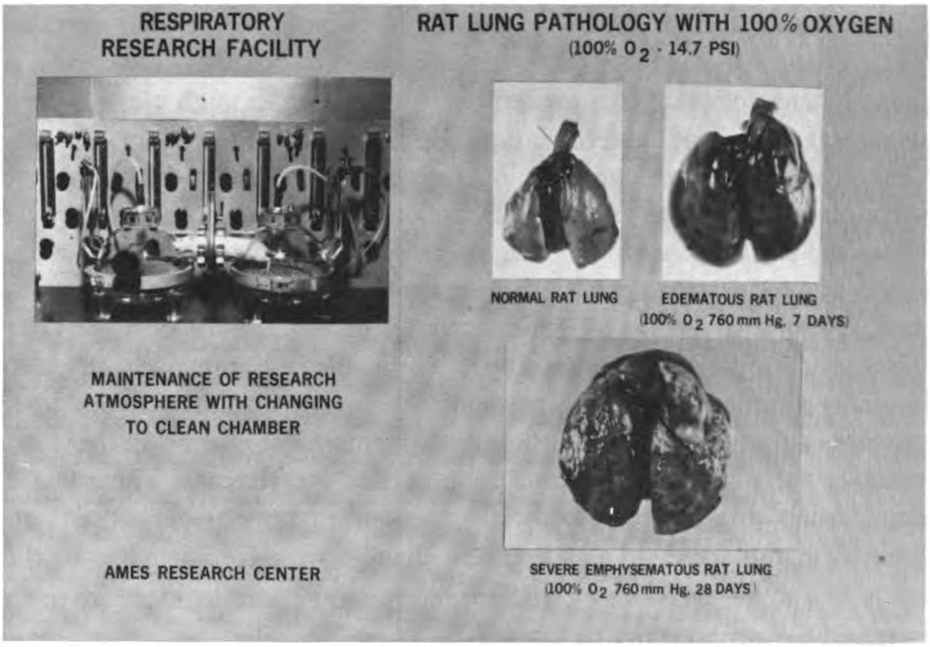
RESPIRATORY RESEARCH FACILITY
MAINTENANCE OF RESEARCH ATMOSPHERE WITH CHANGING TO CLEAN CHAMBER
AMES RESEARCH CENTER
RAT LUNG PATHOLOGY WITH 100% OXYGEN
(100% O2 · 14.7 PSI)
NORMAL RAT LUNG
EDEMATOUS RAT LUNG (100% O2 760 mm Hg. 7 DAYS)
SEVERE EMPHYSEMATOUS RAT LUNG (100% O2 760 mm Hg. 28 DAYS)

FIGURE 21–18. *Respiratory research facility in NASA Ames Research Center (left). Rat lung pathology (right)* (NASA PHOTO).

Atmospheric Particulate Matter

In the weightless environment of a spacecraft, there are two essential differences in the behavior of dust particles. First, sedimentation, which is totally due to gravitational force on the particle, will not occur. Consequently, a considerable range of particles, i.e., those greater than 0.5 and less than ten microns, will be stabilized in the atmosphere. Second, the mean free path of the air molecules will be increased by the reduced barometric pressure. This, in effect, lowers the resistance of the air to particles of all sizes, i.e., increases their mobility, and is important for enhancing diffusion deposition, the dominant factor for deposition of submicronic particles (Morrow, 1966). As part of the Human Factors Systems Program, the Electronics Research Center of NASA has developed an instrument called a nephelometer, or aerosol particle analyzer. This measures the concentration of dust particles in sizes ranging from 0.5 to ten microns, the range of biological interest. In this device, aerosol particles are drawn into a small

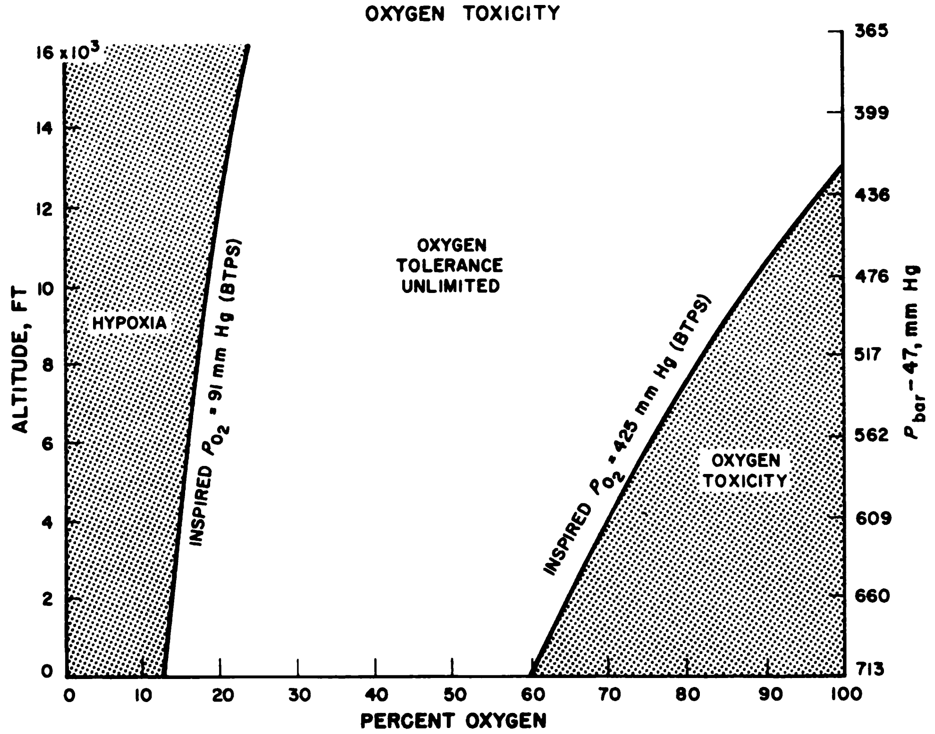

FIGURE 21–19. *Oxygen tolerance in man* (ROTH, 1964; AFTER MULLINAX & BEISCHER).

sampling volume where they are illuminated by a beam of light. Light scattered by a single particle is detected by a photomultiplier tube, resulting in a pulse whose amplitude is a function of particle size. Use of this device yields an accurate indication of particulate concentration and distribution by particle size.

Visual Environment

Many of the requirements of space missions such as Apollo are absolutely dependent on the vision of the astronaut (Jones, Allen, & Parker, 1966). However, the visual environment in which the astronaut will be operating will be different both from that found on earth and in aircraft flight. It is imperative, therefore, that answers be provided concerning the visual requirements of space missions, the visual environment within which the astronaut will be operating, and the effectiveness with which he will be able to perform the required visual tasks. Certain information is available at this time. Much remains to be obtained.

The visual environment to be found in space and during periods of extraterrestrial exploration will be altered considerably from that of earth, primarily as a result of changes in solar illumination and in the atmospheric qualities of the surfaces being explored. Table 21–6 presents available information concerning those parameters of the space environment which will directly affect vision. Note that on the moon the level of solar illumination is approximately one-fourth higher than on earth, the reflectivity of lunar materials is lower, and there is a complete absence of atmospheric light-scattering particles. As a result, some adaptation period may be required before an astronaut can perform visual tasks on the lunar surface with the same proficiency as on earth.

Visual Performance in Space.—The effect of extended residence in space on the efficiency of the visual system is of concern to NASA. As a result, efforts have been undertaken to develop techniques for obtaining precise measures of the visual proficiency of astronauts prior to a mission and again while in flight. In this manner, any subtle changes in visual performance which might occur can be detected and assessed before the problem reaches such magnitude as to affect actual mission performance.

Preflight, inflight, and postflight tests of the visual acuity of Gemini V astronauts were obtained by the Visibility Laboratory of the Scripps Institution of Oceanography (Duntley, Austin, Taylor, & Harris, undated). An Inflight

TABLE 21-6.—*Primary Parameters of the Visual Environment of Space*

	90° Solar illumination	Surface reflectance*		Mean atmos. transmission
Earth	10, 800 ft-c	Ocean	.03	**.70–.80
(Night illumination with full moon = 0.04 ft-c.)		Ground	.15	
		Snow	.80	
EVA (Earth orbit)	12, 700 ft-c	Aluminum	.55	1.00
		Dark paint	.10	
		White paint	.80	
Moon	12, 700 ft-c	Maria	.07	1.00
(Full earth = 1.25 ft-c		Crater wall	.20	
30° phase = 0.80 ft-c				
90° phase = 0.26 ft-c.)				
Mars	7, 600 ft-c	Maria	.17	.80
		Continents	.18	

*Lunar reflectances from Conklin (1965) and NASA (1966). Mars reflectances from Haines (1965).
**Function of diameter and distribution of scattering particles.

Vision Tester was used, which was a small, self-contained, binocular optical device containing a transilluminated array of high- and low-contrast rectangles. Astronauts judged the orientation of each rectangle and indicated their response to this by punching holes in a record card. With this technique, astronauts were able to test themselves with their responses stored for later evaluation.

As a second part of the Gemini V program to measure visual capability, large rectangular patterns were prepared at ground sites in Texas and in Australia. The task of the astronauts was to report the orientation of the rectangles. Provision was made for changing rectangle orientations between passes and for adjusting their size in accordance with anticipated slant range, solar elevation, and the visual performance of the astronauts on preceding passes.

Results of the Gemini V program indicated that the visual performance of the astronauts neither degraded nor improved during the eight-day mission. Although operational difficulties and meteorological conditions interfered with the program to view the ground panels, success was achieved on one pass. Results appear to confirm that the visual performance of the astronauts during space flight was within the range predicted by measures of their visual acuity combined with environmental optical data.

Protection of Vision.—Due to the high levels of visible radiation in space, there is a requirement for functional protection for the eye. As indicated in table 21-6, brushed aluminum with a reflectivity of .55 will have a brightness of approximately 7,000 foot-Lamberts under direct solar illumination. Inasmuch as the viewing of large objects with a brightness in excess of 1,000 foot-Lamberts produces visual discomfort, there may be a requirement for a protective visor with a transmission of 15 percent or less if extravehicular maintenance activities are to be carried out efficiently.

There is also a danger of permanent injury to the visual system during space flight. Excessive levels of visible or infrared energy are easily sensed by the human and thus provide an indication of dangerous exposure. However, the human possesses no means of detecting ultraviolet radiation. Consequently, severely damaging exposures may be suffered without awareness. It is critical that astronauts be protected against such exposure.

Of the near ultraviolet radiation, that which is most damaging to the eye, 57 percent is absorbed by the atmosphere of the earth (Schmidt, 1962). This compares with an absorption of less than 15 percent for visible radiation. However, it must be noted that the above measures are for vertical incidence and clear air conditions. Thus, under normal conditions in which the sun is not directly overhead, and an atmosphere haze condition exists, and individuals do not look directly toward the sun, the intensity of the ultraviolet energy received is much less than 43 percent of that found outside the earth's atmosphere. The UV protection problem in space thus is more serious than on earth and may require the use of special visor systems which attenuate virtually all of this energy. Present visors, which transmit only one to four percent of this energy, have been found adequate for short-term missions. Their adequacy for long-duration missions remains under investigation.

LIFE SUPPORT SYSTEMS

There is one broad problem area which has been mentioned as one of the most important to which medical science can contribute as preparations are made for deep space journeys (Jones, 1965). This is the problem of developing a true closed ecological system. It is one which is under intensive investigation in OART's Human Factors Systems Program.

The Mercury, Gemini, and Apollo flights used, or will use, life support systems of the "open" type. Such flights can stay in space only until the stored supplies are consumed. This of course limits the effective period of space flight inasmuch as weight problems could preclude the storing of large quantities of consumable items. Since these first flights, systems have been developed to reuse some supplies, especially water and oxygen. The ultimate research goal, however, is a true closed ecological system. A spacecraft in such a system will be balanced so that all supplies are used and replenished at the same rate in a safe and satisfactory environment. Figure 21–20 illustrates the weight benefits to be derived from a system which is regenerative in all aspects except food. It can be seen that for a 600-day mission, a system using stored supplies will require approximately 13,000 pounds per man of materials. A regenerative system will require only about 3,000 pounds per man. The benefits of regeneration of supplies are obvious.

The challenge and difficulties involved in the development of a closed ecological system are tremendous. However, the medical benefit of the technological advances which will occur as this system is developed will extend to more than just space flight. As the population of the world in-

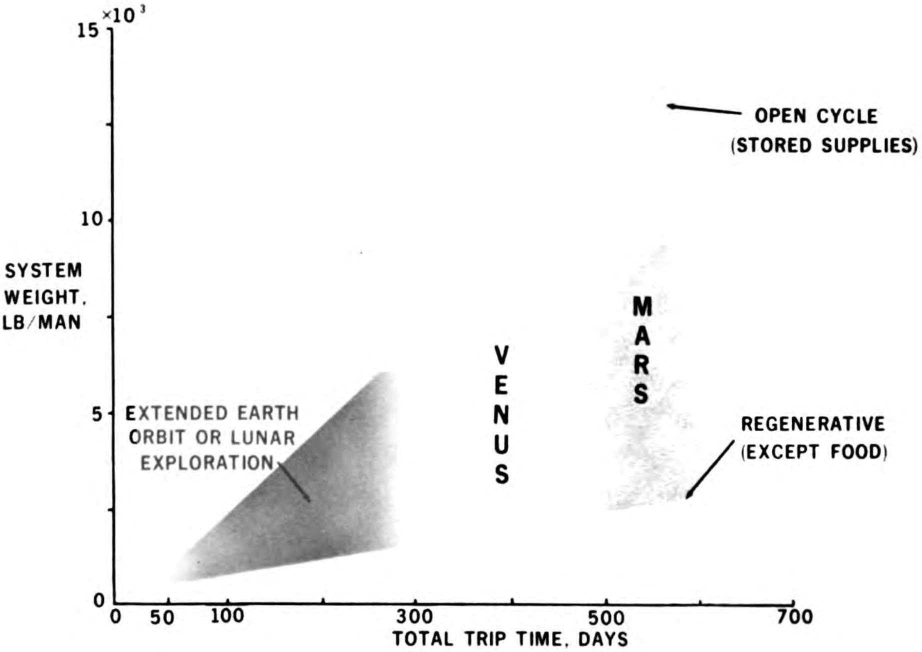

FIGURE 21–20. *Weight benefits to be derived from a regenerative (except for food) life support system* (NASA PHOTO).

creases, and food and water supplies become more precious, this information may be of tremendous value in insuring adequate sustenance for all persons.

Oxygen Reclamation

One of the most important life support concerns is that of the cabin air supply, whether it be of the open or closed type. Cabin transport systems such as jet transports use large amounts of purge air to remove carbon dioxide, trace contaminants, moisture, and odors. A manned planetary or long-term, earth-orbiting spacecraft cannot afford to carry such a system and must therefore rely on a chemical processing system to remove undesirable elements and possibly to regenerate oxygen and water. This system, in addition to performing its normal functions, must be capable of removing trace contaminants or toxic substances generated by the crew, the spacecraft, and the equipment.

The most advanced systems presently built for oxygen recovery require three separate processes involving absorption and reduction of carbon dioxide, followed by electrolysis of water. Technology research is required on new concepts which will reduce these many steps to one electrochemical process. This will permit a continuous flow system relatively unaffected by cabin water vapor. In addition, it will permit simpler control and fewer components which will increase reliability. Similar research is required in the areas of trace contaminant control and removal of toxic products evolved from equipment.

An example of the research being conducted by NASA to develop improved methods for oxygen reclamation is provided by the Langley Research Center program which is studying both solid and molten electrolyte methods. Either of these methods might replace, as a single unit, the silica gel, molecular sieve, Bosch reactor, and electrolysis components, as shown in figure 21–21, of the present Langley space flight simulator. The composition of the solid electrolyte is being studied with the objective of lowering operating temperatures and achieving the associated longer operating life.

The molten carbonate concept of electrolysis is inherently more efficient than the solid electrolyte in recovering oxygen from carbon dioxide, but it presents the problem of carbon removal from the electrolyte under weightless conditions. Different methods of carbon removal are being investigated. They include direct removal by withdrawing the electrode on which the carbon is formed, as well as centrifugation removal.

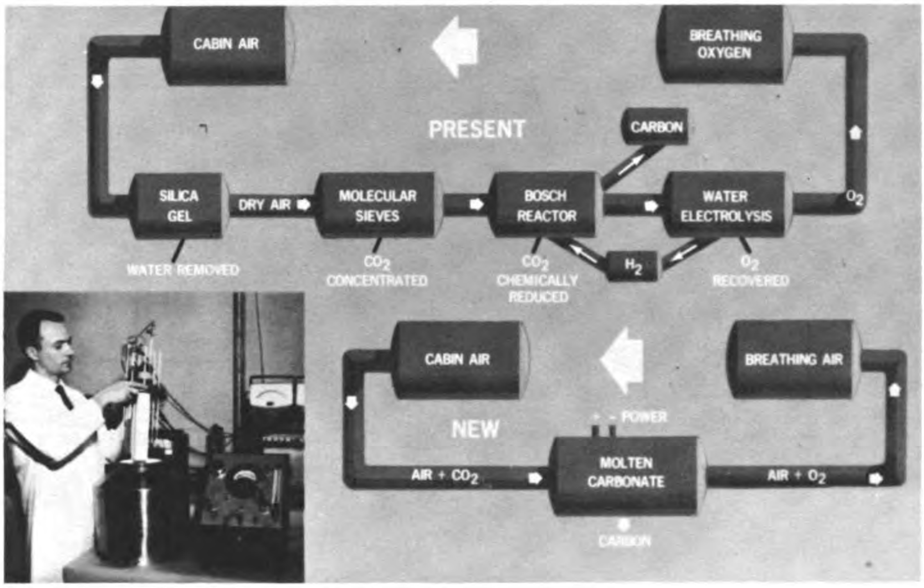

FIGURE 21–21. *NASA Langley Research Center oxygen reclamation system* (NASA PHOTO).

Water electrolysis processes are used in some current life support systems for oxygen recovery. Conventional electrolysis units depend on gravity to separate the gases from the liquids. In the zero-gravity conditions of space, techniques which do not require a gravity field must be utilized or an artificial gravity field provided.

A phosphoric acid electrolyte cell, suitable for operation under zero-gravity conditions, has been developed for the Ames Research Center. This cell, shown in figure 21–22, uses a vapor-feed principle since electrolysis of man's exhaled and perspired moisture will supply his required two pounds of oxygen per day. When the vapor in the air is electrolyzed, an auxiliary function of cabin humidity control also is served. In all tests to date, these cells have performed satisfactorily with no apparent change in current voltage characteristics when operated under conditions of humidity and temperature simulating the space vehicle cabin. It is estimated that this cell will weigh ten pounds per man (fixed weight) and require 287 watts per man to operate. This cell represents one of the most advanced and most reliable vapor-feed electrolysis cells for use in zero gravity.

Also under study is another vapor-feed electrolysis cell, shown in figure 21–23, which uses sulphuric acid as the electrolyte. This cell is more efficient

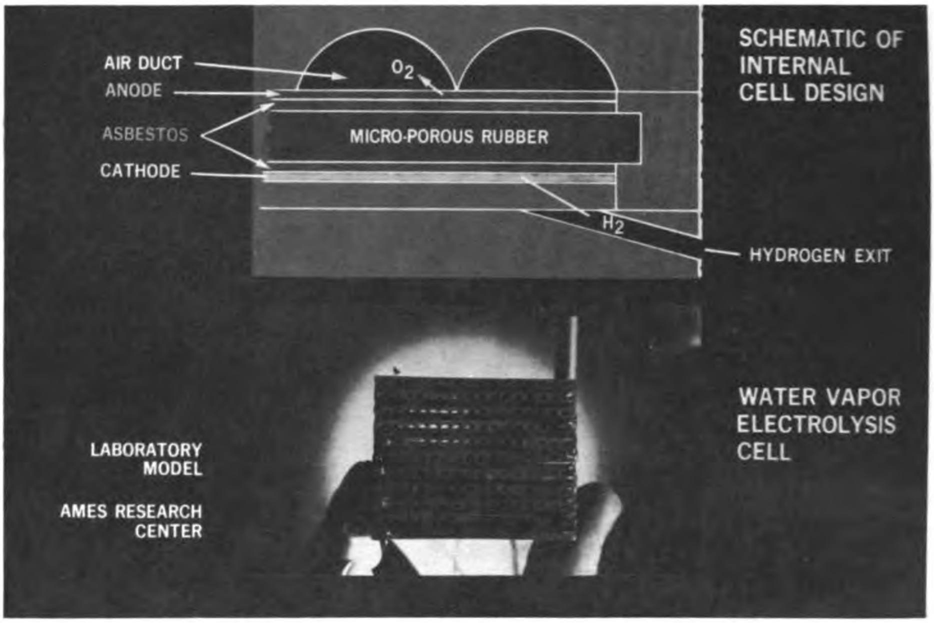

FIGURE 21–22. *Laboratory model of water vapor electrolysis cell developed by NASA Ames Research Center* (NASA PHOTO).

than that using phosphoric acid. However, it has not been carried to an advanced state of development. There is a possibility of contaminant formation at the electrodes of the sulphuric acid cell due to breakdown of the electrolyte. In order to probe this possibility and to develop a basis for new oxygen-reclamation concepts, NASA is supporting investigations of the basic electrochemical principles involved in electrolysis.

Trace Contaminants

In order to conserve atmospheric materials on extended manned missions, there is a requirement for near-zero leakage from a space cabin. Under these conditions, the buildup of contaminants from equipment and from man himself can become a serious problem. Developing an understanding of man's tolerance to various atmospheric trace materials for long periods of time is a primary requirement. A second requirement involves prevention of contamination through the selection of proper spacecraft materials. Both of these requirements, as well as detection and removal concepts, are receiving considerable study in NASA.

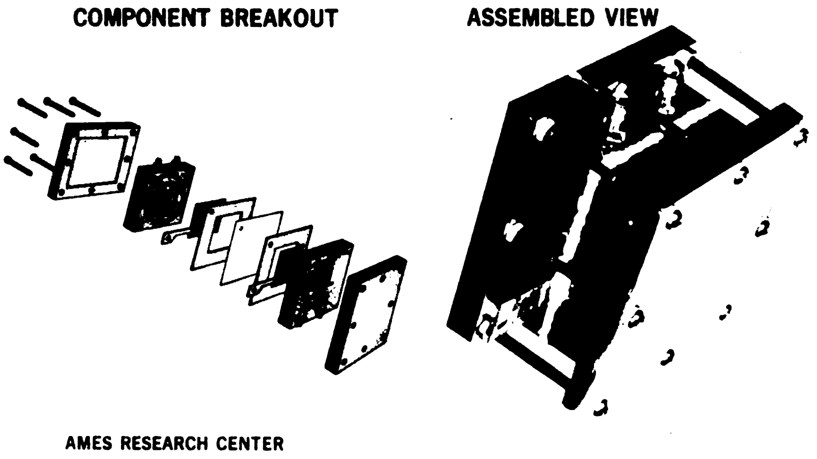

FIGURE 21–23. *Sulphuric acid electrolysis cell* (NASA AMES RESEARCH CENTER PHOTO).

Conventionally, activated carbon is used to remove aerosols, while a catalytic burner is used to remove contaminants such as carbon monoxide. Studies at the Langley Research Center are directed toward means of reactivation of the carbon and improvement of the catalyst. Langley has installed a chamber, shown in figure 21–24, for testing the removal effectiveness of new catalysts. Gas chromatographs, mass spectrometers, and other instruments are used to determine the concentration of the contaminants in the chamber.

A new technique for removal of contaminants, involving freezing, currently is under study. The different contaminants which can be removed by freezing will be determined, along with the temperatures required, and the other necessary engineering parameters. It may be possible to remove all contaminants by this freezing procedure. This would eliminate or greatly simplify the identification problem. For identification of contaminants, improved gas chromatographs and mass spectrometers capable of installation in a space vehicle are being investigated, along with other possible sensing techniques. The ability to sense the complete spectrum of the spacecraft contaminants has not yet been achieved.

Two-Gas Atmospheres

As mission durations increase to include manned planetary flight, extended earth orbital, or long lunar surface missions, the present 100 percent oxygen

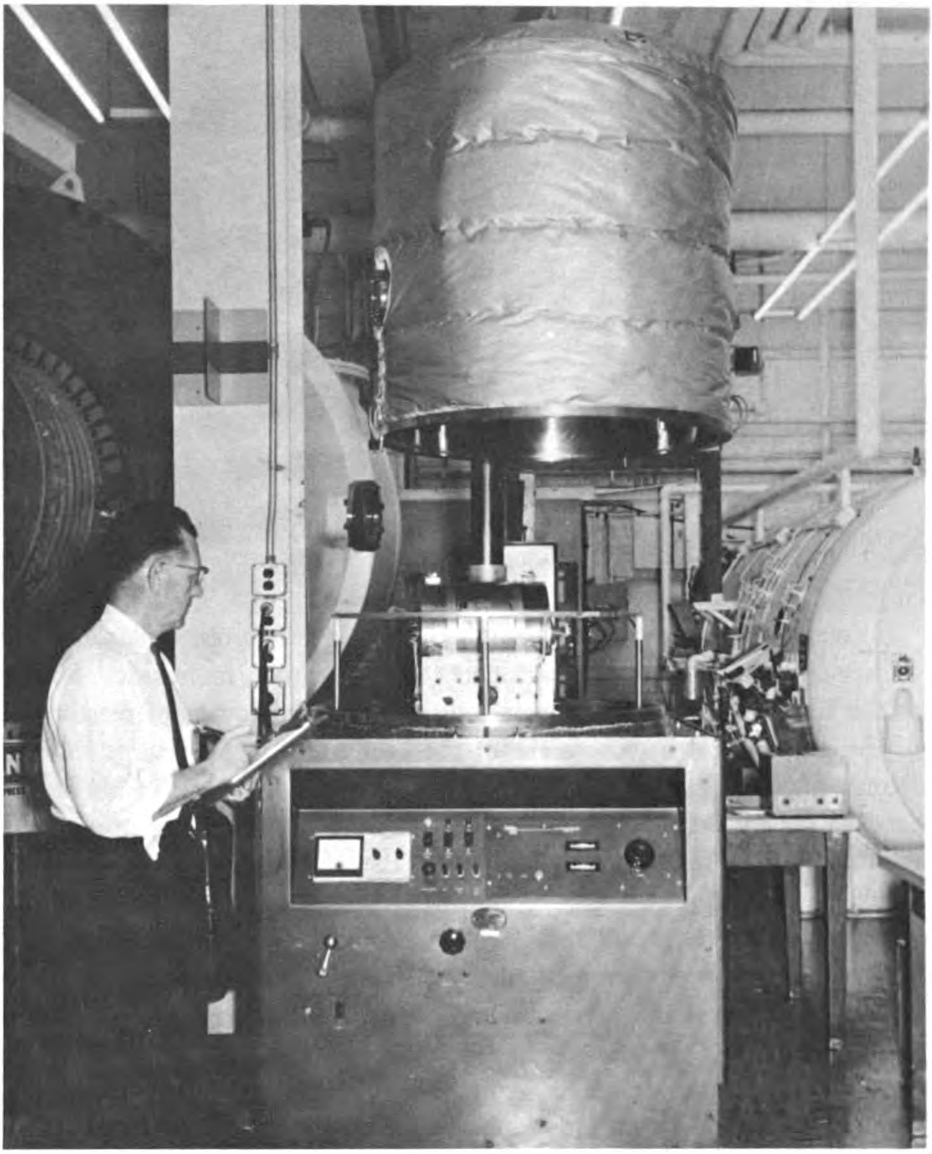

FIGURE 21–24. *Trace contaminant research facility at NASA Langley Research Center* (NASA PHOTO).

systems will be modified to use a diluent such as nitrogen or helium. Such systems will require sensors for oxygen, carbon dioxide, water vapor, and the inert gas. To meet the technological need for flight sensors, the Langley Research Center is investigating two methods of sensing these parameters.

One concept uses a mass spectrometer especially designed to separate and measure the four gases. The second approach uses ultraviolet to measure oxygen and water vapor, and infrared to measure carbon dioxide partial pressure. A total pressure sensor is used in combination with the other measurements to determine nitrogen.

Studies of simpler but more advanced sensing methods also have been started. One is a solid electrolyte oxygen sensor. Emphasis is on the reduction of power and the amount of reference gas needed. The size of the unit is indicated on the right side of figure 21–25. Oxygen ions pass through the solid electrolyte from a reference sample of 100 percent oxygen to the measured gas stream. The voltage developed by this passage indicates the oxygen level. Another advanced concept shown in figure 21–25 is the use of a thin film technique. The amount of oxygen in the cabin air causes the resistance of a gold film to change, thereby giving a measurement of the oxygen present. The film is only 500 angstroms thick. The development of this technique is in its early stage but shows great promise.

Food Supply

Total food cycling is considerably beyond the state of today's technology and science. Consequently, primary research efforts today are directed toward reducing the volume of stored food while maintaining palatability for morale purposes. The Army Natick Laboratories are studying advanced

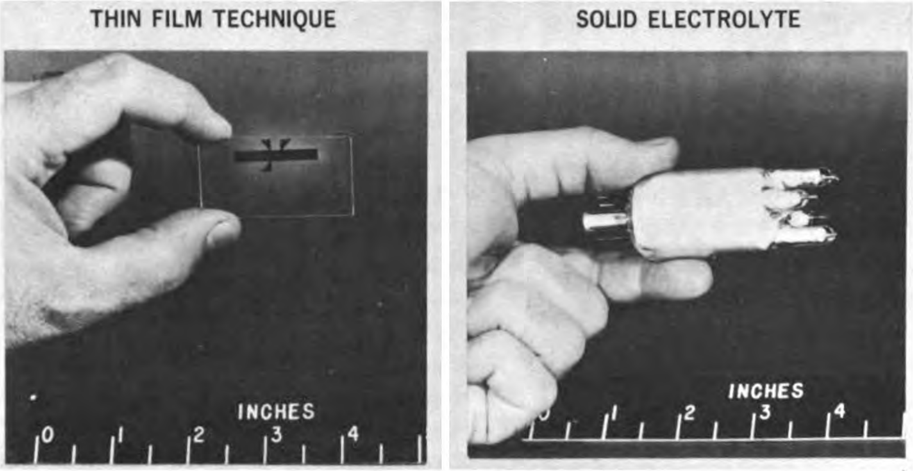

FIGURE 21–25. *Oxygen-sensing methods under study* (NASA PHOTO).

dehydration techniques, considering the compromise between reduced weight versus nutrition and palatability. To reduce dry volume, high compression of dehydrated foods is being investigated. One attractive approach uses the concept of a limited variety of precooked food in the form of bars, to which can be added seasoning, sauces, and condiments, in the form of dry sheets, thereby achieving a menu variety. The advantage of this approach is illustrated by the fact that five compressed bars alone would give a choice of five meals such as: (1) beef, (2) chicken, (3) rice, (4) peas and carrots, and (5) potatoes. The additional sheets of sauces, gravies, and seasonings will insure the availability of a number of different variations of this menu. Other compressed products include peas and shrimp which have been dried and compressed to one-third of their original volume, as shown in figure 21–26. Upon rehydration, they are returned to approximately their original size with taste and texture exceeding the flavor of available conventional freeze-dried food. Such techniques permit the storage of a 45-day solid food supply in the same volume presently required for a 15-day food supply.

A still more difficult task confronting life support systems engineers is that of totally closing the food cycle. The objective here is to develop an

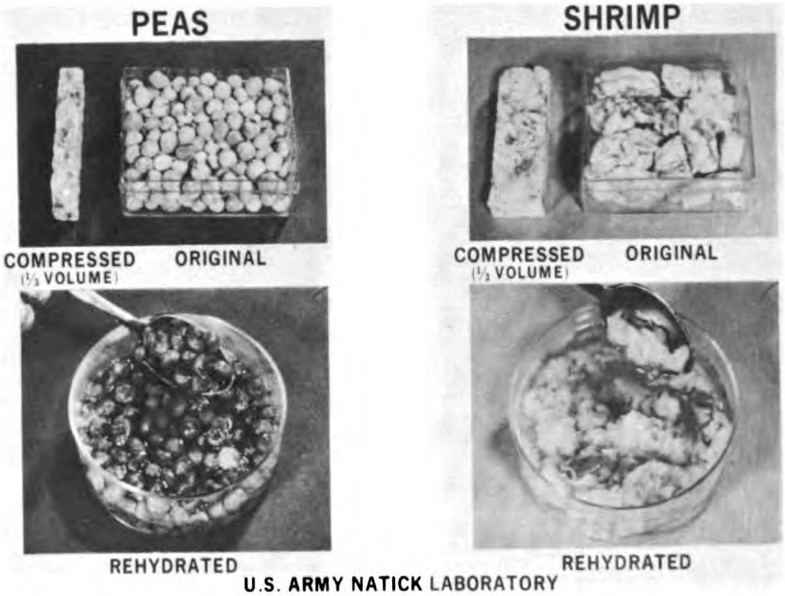

FIGURE 21–26. *Compressed dehydrated foods* (NASA PHOTO).

ecological system which will support human life for periods of one to three years without the need for resupply. Probably the first system will use physical-chemical processes in addition to bioregenerative techniques. Figure 21–27 depicts the operation of such a system. Physical-chemical food synthesis may provide simple sugars or carbohydrates by synthesis, and amino acids or proteins by biological means. An activated sludge waste processing unit would convert urine, feces, and food residues to nutrients for the biological reactor, Hydrogenomonas bacteria. The Hydrogenomonas cells would be harvested and processed for use as food, thereby providing protein. The physical-chemical carbohydrate synthesis unit would use carbon dioxide as the raw material to make a mixture of sugars. The water formed in the Hydrogenomonas process and in the carbon dioxide reduction process will provide cabin water and will be electrolyzed to obtain the required oxygen. In this manner, oxygen, water, carbohydrates, and protein can be supplied. Minor supplements of minerals, vitamins, and, possibly, fats will be required. It is not expected that the complete technology for this system can be accomplished before 1975.

Life Support System Testing

Extensive testing will be required, under simulated space conditions, to assess the performance of a total life support system and to identify the

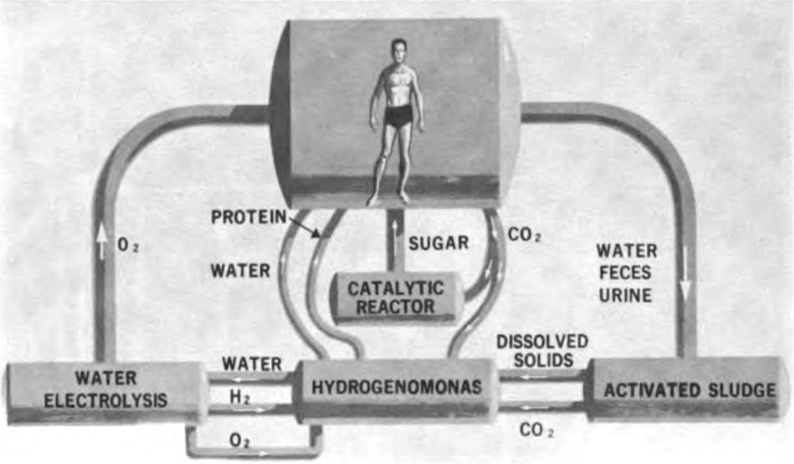

FIGURE 21–27. *Future physicochemical-bioregeneration life support system under development at NASA Ames Research Center* (NASA PHOTO).

related maintenance requirements. A system allowing lengthy manned simulation studies has been installed at the Langley Research Center, as shown in figure 21–28. Living spaces and facilities, as well as components of the atmosphere subsystems, are shown in figure 21–29.

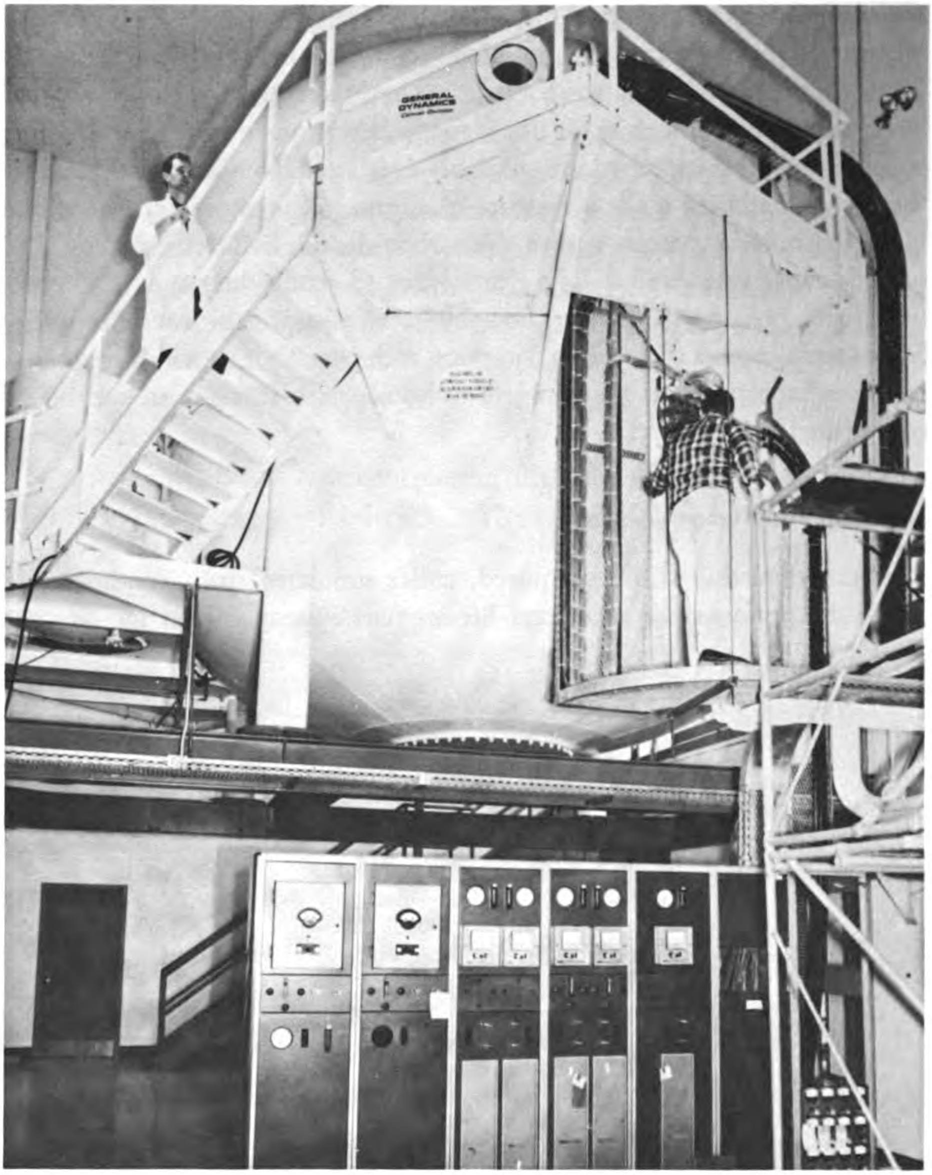

FIGURE 21–28. *Recycling life support system installed at NASA Langley Research Center* (NASA PHOTO).

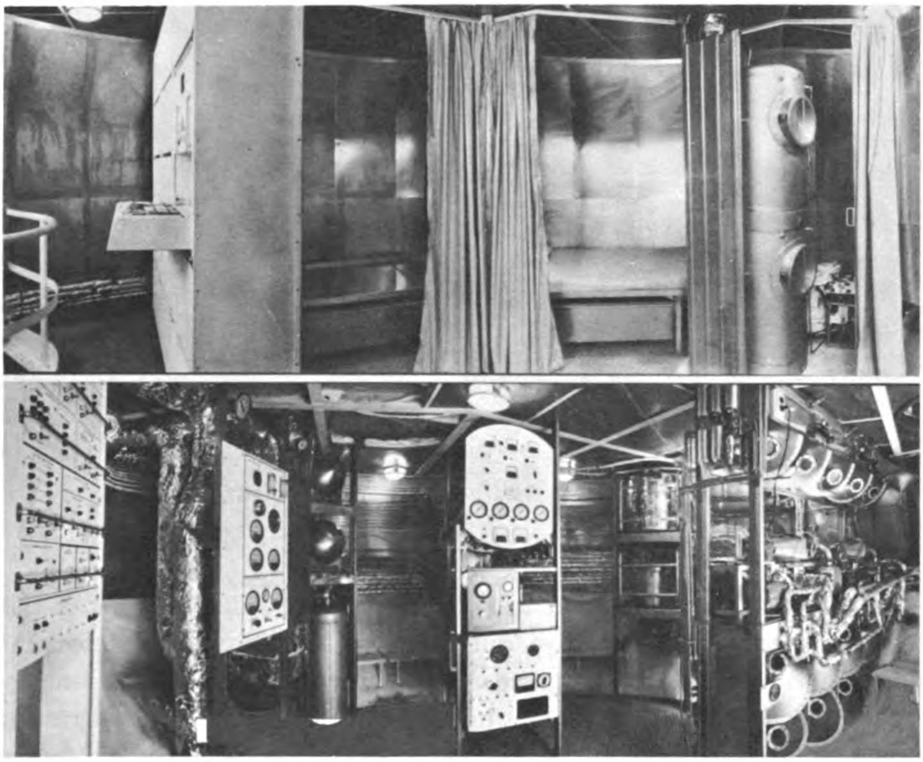

FIGURE 21–29. *Interior of simulated vehicle for life support tests at NASA Langley Research Center* (NASA PHOTO).

Use of the Langley facility will allow system-man interactions to be determined under actual test with varying workloads. In tests to date, techniques have been developed for using waste heat from other systems as direct energy for the life support regeneration processes. The capacity of the laboratory water electrolysis units has been increased to match the crew's requirement for oxygen. Water reclamation, carbon handling, and trace contaminant control processes are being operated in conjunction with each other in the closed cabin. Tests such as these will supply needed data for mission analyses by determining interrelated performance requirements, as well as the actual man-hours required for operation of the system. Functions which should be automated can be analyzed and compared with the possible reduction of reliability due to increased complexity. Interactions among the various subsystems can be determined along with their individual and collective interfaces with the power system. The final result will be a total validation of the interactions among various components of the life support system during an extended space flight simulation.

PROTECTIVE SYSTEMS FOR SPACE MISSIONS

The NASA program for the development of spacesuits has its roots in the Navy full pressure suit program. In 1959, a slightly modified version of the Navy MK IV full pressure suit was selected for use by Project Mercury astronauts. Gemini suits were an adaptation of an Air Force suit. The Apollo suits were developed especially for NASA purposes. Many of the NASA personnel directing this program, however, were formerly associated with the U.S. Navy and U.S. Air Force full pressure suit efforts. It is not surprising, then, that many of the protective concepts embodied in pressure suits remain similar.

The provision of the technology required for the development of life support systems and protective suits for long-duration missions remains one of NASA's most critical needs. Gemini and Apollo systems are designed for two-week flights. Inasmuch as all life support material can be stored for missions of this duration, suits for these missions are not as complex as will be those for longer missions, in which portable, regenerative systems must be incorporated.

Representative of present Biotechnology Advanced Research Program efforts in protective systems is the advanced spacesuit for more extended lunar and extravehicular operations, the anthropometric RX-2A suit, shown in figures 21-30 and 21-31. This is being developed for OART by the Manned Spacecraft Center. Environmental control and life support are provided by the back pack shown on the back of the suit. The suit uses "constant volume" joints which have essentially zero resistance to movement and thus permits maximum mobility both in free space and on the lunar surface. The suit represents a breakthrough in permitting maximum mobility with low energy expenditure. Forces required for movement are now so low the wearer is scarcely aware he is pressurized. For this reason, it has been necessary to provide interlocks to prevent inadvertent opening of the suit when it is pressurized. The suit is made of a laminated, honeycomb, aluminum structure and will potentially permit the use of a ten-psi differential pressure with a two-gas atmosphere. Inherent in the construction of this hard suit is the capability for greater protection against radiation and micro-

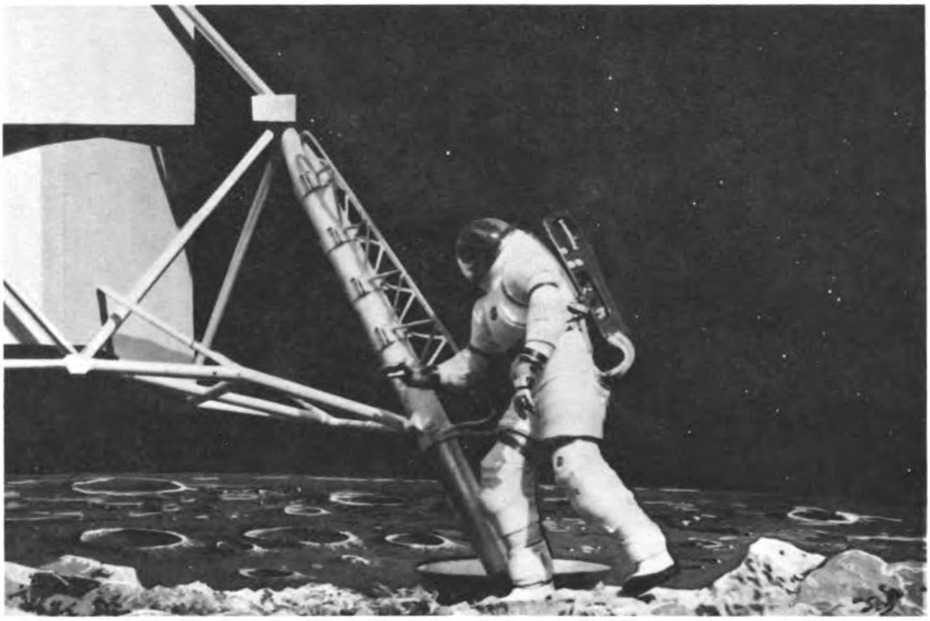

FIGURE 21–30. *RX–2A advanced spacesuit (hard suit)* (NASA PHOTO).

meteoroids than present suits, but with reduced bulk. Leakage has been reduced to less than one-half that experienced with earlier suits.

An in-house development effort at the Ames Research Center has produced a first version of another anthropometric suit, called the AX suit. It embodies a number of unique concepts which make this suit a logical successor to the RX–2A and the one which may be used on Mars missions. One major difference with the AX suit is the rotating pseudoconic joint, used to provide movement in the arms and legs. The operation of these can be seen in figure 21–32. The AX suit also provides places in the glass fiber construction for installation of either insulating material or a liquid coolant to provide protection against extreme thermal environments. This suit also uses a bubble-dome helmet, as does the RX–2A, in order to provide unrestricted visual fields during extravehicular activity. The projected leakage rate, movement torques, and total suit weight place the AX in a favorable position as the likely garment for future long-duration missions.

A nonanthropometric suit development is represented by the inflated sphere shown in figure 21–33. In this version, the astronaut can move by simply walking inside the sphere. On one side, are fitted arms and legs

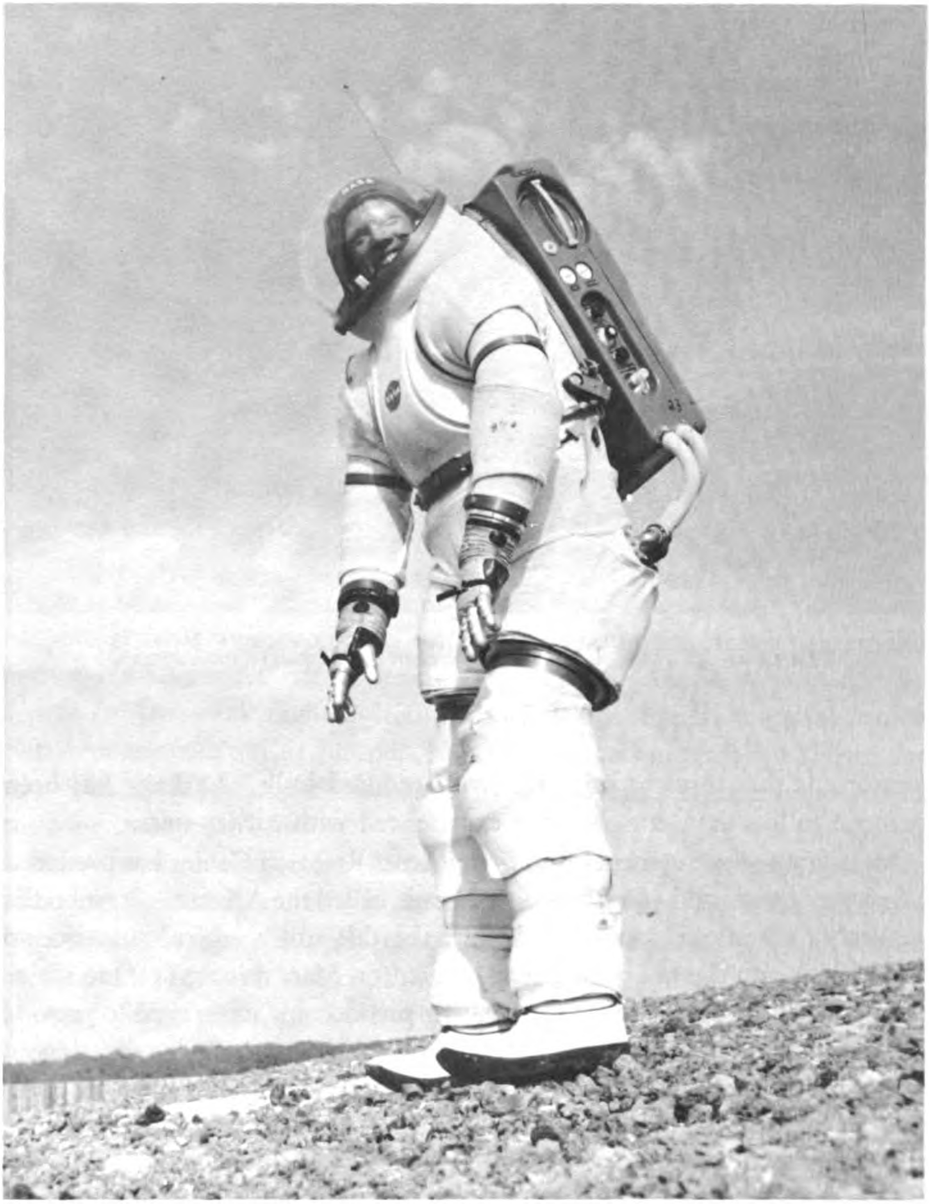

FIGURE 21–31. *RX–2A advanced spacesuit* (NASA PHOTO).

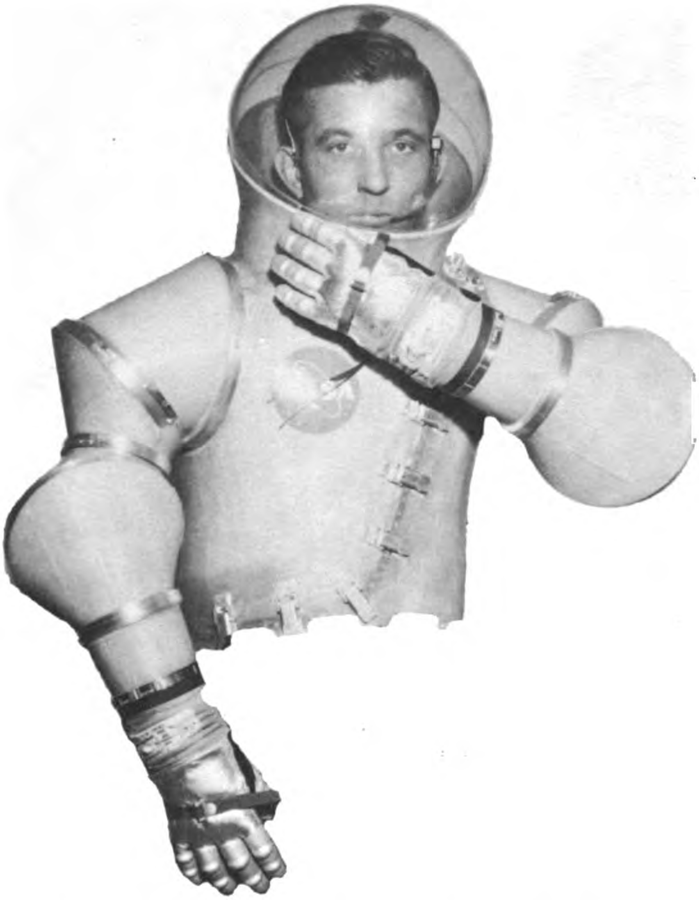

FIGURE 21–32. *Advanced AX spacesuit* (NASA PHOTO).

so that the astronaut in the 1/6 gravity environment of the moon could easily walk using the legs and supporting the sphere on his back. A pass-through lock permits him to bring an article inside the sphere (figure 21–34) where he can perform delicate manipulations using his bare hands. This particular development effort represents only a feasibility study at this point in time.

ADVANCED CONCEPTS RESEARCH

Certain of the research currently supported in the Human Factors Systems Program of NASA is directed toward what is termed "advanced concepts." It is felt that such research is essential if man is to be ready to solve the next

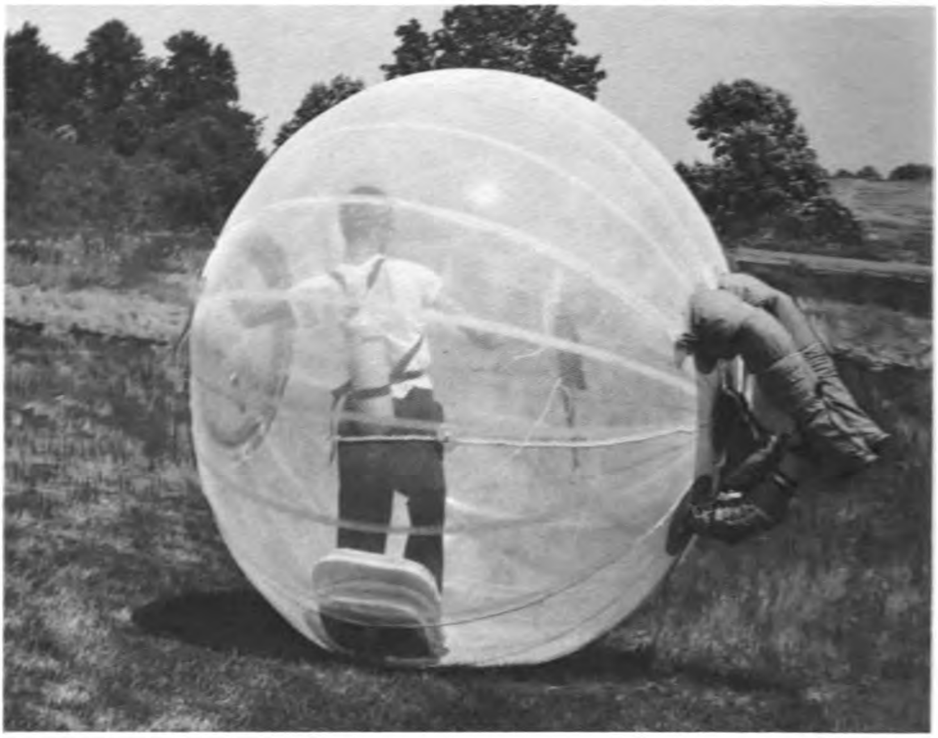

FIGURE 21–33. *Nonanthropometric spacesuit* (NASA PHOTO).

generation of problems after those currently confronting him (Jones, 1965). In life sciences research, many of these problems involve ways of further integrating man and his spacecraft. In recent years, several interesting possibilities have arisen.

The precision control of high-speed aircraft has, under certain circumstances, already taxed to the limit the ability of man to coordinate sensory perception with motor response. The task of controlling the complex spacecraft of the future could easily exceed man's ability. One limiting factor is the time required for sensory cues to be transmitted to the brain, translated into a motor command, and retransmitted to the hands and feet in order to make the necessary response. Were a direct link to be established between the eye and the mechanical system to be controlled, this time limitation could be circumvented. The feasibility of building a device which could sense eye movements and utilize these movements to generate control systems is under study by NASA. The device would follow the motion of the eye by tracking a beam of light reflected from the cornea. The resultant motion

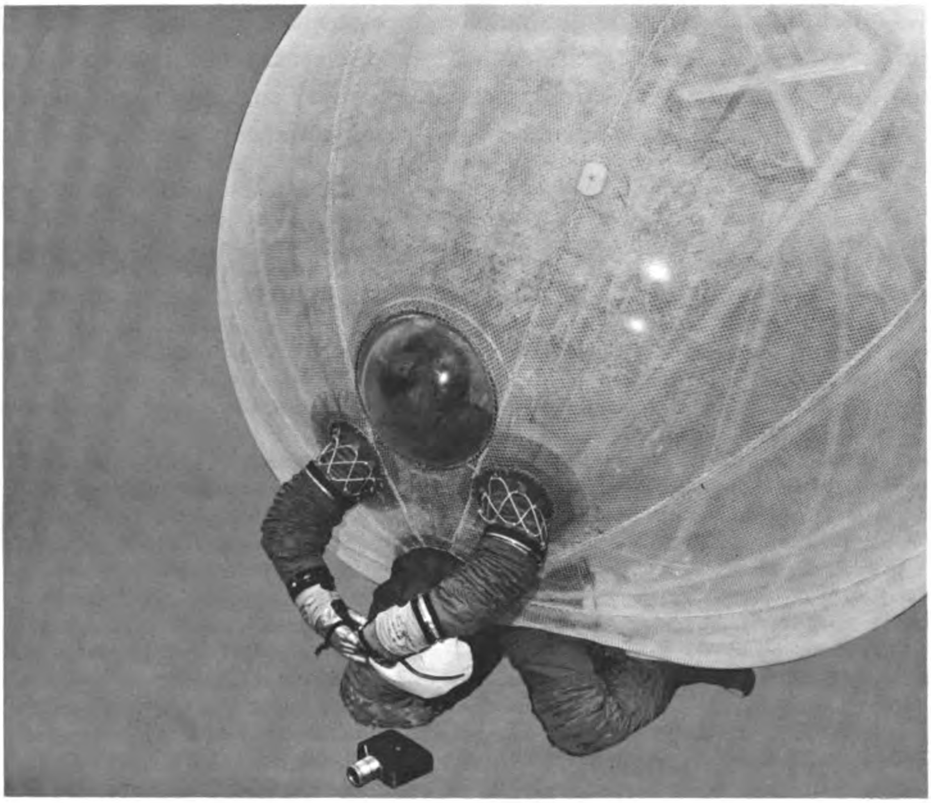

FIGURE 21–34. *Object manipulation while wearing the nonanthropometric spacesuit* (NASA PHOTO).

of the light beam would be converted into electronic signals. These signals could then be used to command certain control operations. With such a device, the astronaut could maneuver his spacecraft using eye movement alone.

A second possibility, a step beyond the first, is the direct transmission of control signals from man to machine by nerve impulses. With such a technique, machine systems could be controlled directly by the brain and nervous system in the same manner that movements of man's limbs are controlled. Current research is defining the complete electromagnetic character of neuronal impulses and, if successful, will provide a starting point for later studies of control applications. Results of these studies will have great impact on the control of vehicles on earth as well as in space. In addition, such control systems offer great hope for individuals who might have suffered partial or total loss of their motor capability.

Opportunities exist for Flight Surgeons to participate in advanced research programs such as those described above. Research such as this, which strives to push the boundaries of current technology, is very challenging and, when successful, aids our space program directly and ultimately provides benefit to all of mankind.

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